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Report of a Series of Warfarin Poisonings

Neomycin Sulfate in Veterinary Dermatology and Ophthalmology

Canine Pathology: An Historical Essay

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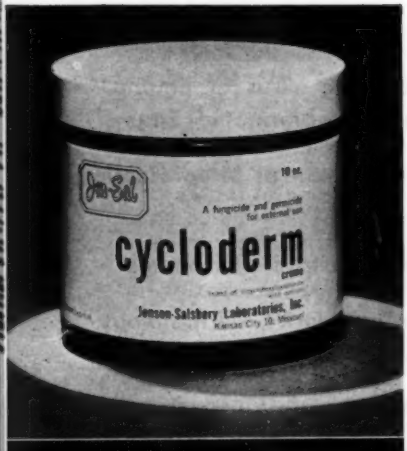

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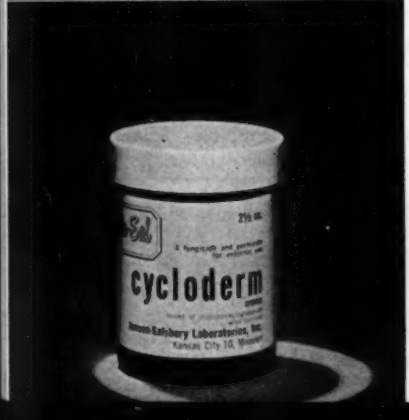

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Influence Of Protein On The Milk Fever Complex (A Four Year Study)

Part I: Milk Fever

R. F. VIGUE, D.V.M., Springvale, Maine

THE cause of milk fever is generally given as an acute calcium deficiency resulting from the sudden reduction of blood calcium at the onset of lactation. This, and the fact that the disease is primarily one affecting excellent dairy cows that are usually fitted well at the time of calving, are the only two definite conclusions that can be reached on review of the voluminous literature on the subject. Despite usual prompt clinical response to calcium therapy in treatment of milk fever, liberal feeding of various calcium salts has not reduced incidence. The calcium theory has been rejected by many practicing veterinarians, as well as dairymen. It is now conceded by a host of workers that the fundamental etiology of the disease remains unknown, which opinion also has been expressed by prominent clinicians.

Review of Literature

Greig,[1,2] who first advanced the calcium hypothesis in 1925, expressed doubt that the disturbance in calcium metabolism alone was responsible for the anomaly referred to as milk fever. In explaining his theory, he stated that the sudden withdrawal of this mineral from the blood could not in itself be regarded as the cause, because if this were so, every heavy milking cow would be subject to the disease. For this reason, he believed that some other factor must act as a predisposing cause and suggested parathyroid dysfunction. This idea was based on the fact that the parathyroid hormone (parathormone) is known to regulate total serum calcium and control the calcium-phosphorus balance. Reduction of blood calcium has long been known to result in excitability, muscular contractions, and a condition simulating epilepsy. Unfortunately, the test of time has proved that parathormone has had very little influence in treatment or prevention of the disease. Recent developments in calcium metabolism have revealed that total serum calcium is dependent upon the protein nutritional state of the animal, and this will be discussed subsequently.

After observing 300 cases of milk fever complex, including ketosis, grass tetany, and toxemia of pregnancy, Barker[3] concluded that milk fever is much more than a simple hypocalcemia. Udall[4] has written that the increasing frequency of milk fever, and of deaths or relapses following the usual treatment, suggest that commonly accepted theories of its cause are still incomplete. Failures in present therapy have led Jones[5] to report that there is far from total agreement that milk fever is due exclusively to calcium deficiency. He believes that the cause is more basic and concludes that relapses should be expected, inasmuch as our present therapy is aimed at a symptom rather than at the cause. Roberts[6] has also been concerned with recurrence of milk fever symptoms in cows treated with calcium over a period of two to five days after calving, with failures of response to further treatment, and eventual death. Disappointment in present methods of handling this disease has prompted Bowen[7] to call the calcium theory a myth. He reports that the more experience he has with various manifestations of milk fever, the less satisfied he is with present concepts concerning it.

Etiology of Protein Imbalance

The writer has observed an increasing number of calcium refractory cases of milk fever, and has had to rely heavily upon udder inflation to supplement calcium infusion therapy. Use of antibiotics or sulfonamides in the udder prior to inflation eliminates the hazard of mastitis, provided other precautions are taken with equipment. Prompt response to udder inflation of cows having received up to six therapeutic doses of calcium and other minerals without apparent beneficial effect, suggests that a substance present in the milk is responsible for the recovery when it is returned to the blood stream. That substance is protein. Table 1[8] indicates that first milk, colostrum, is about one-fifth protein.

Recent knowledge of calcium and protein

metabolism has established a very close relationship between the two. In milk, the very active element calcium does not occur freely,

TABLE 1. Composition of Milk*

	Water	Fat	Protein	Lactose	Ash
Normal milk	87.25	3.80	3.50	4.80	0.65
Colostrum	71.69	3.37	20.68	2.48	1.78

*Eckels, Combs, and Macy

but is found conjugated to a protein molecule complex. These molecules are called phosphoproteins and occur chiefly as caseinogen, the principal protein of milk. When caseinogen is subjected to the action of weak alkalies, it yields calcium caseinate[9]. It now becomes apparent that when udder inflation is employed, regardless of previous therapy, not only calcium but a very large amount of protein is returned to the cow's circulation. The globulins of milk and blood are identical,[10] and the other milk proteins are also derived from blood protein. The amount returned to the bloodstream has never been determined, but it is probably the most important single factor contributing to recovery in calcium-resistant cows.

The protein mixture of colostrum has long been known to contain a very special amino acid mixture that cannot be matched by food protein. It must come from the protein reserves of the cow and these are not large. Tolly[10] has shown that milk proteins are derived from blood proteins and the latter, of course, come from dietary sources. Furthermore, serum protein determinations in many high producing dairy cows during the dry period have been considerably lower than the normal range. Synthesis of protein in the cow, whether measured as net gain of protein or as turnover, is slow when this process is compared with many ordinary chemical reactions. The protein molecule can only be synthesized when all the amino acids which go into making up the molecule are present at the time.

Borsook[11] has reported that there is apparently no mechanism whereby amino acids can be stored individually or in polypeptide groups. This has been proved in experiments in which two incomplete but supplementary mixtures of amino acids or proteins failed to give nitrogen retention when separated from one another by a few hours, but did so when fed simultaneously. In his experiments, Geiger[12] also found that the process of protein synthesis requires that all the amino acids which go into formation of a protein molecule be present at the same time.

Importance of Protein Quality

Dietary proteins vary greatly with respect to being utilized and retained for the synthesis of tissue protein. This characteristic of food protein, referred to as nutritive value, is dependent for the most part upon the amino acid composition. Essential amino acids must be present in adequate amounts for a protein to be well utilized. Small changes in composition are reflected in changes in nutritive value, even though all essential elements are supplied in some quantity. Therefore, a protein deficient in a specific amino acid is limited in its utilization to the extent of that deficiency.

Feeding more of a ration low in one or more essential amino acids that are not supplied by other dietary sources does no good. This will explain failures in the past of several experiments in which increasing the quantity of the same grain ration, lacking a well balanced amino acid composition, failed to reduce incidence of milk fever or ketosis. It is also known that proteins having different patterns of amino acid composition supplement one another, so that the nutritive value of the mixture is higher than the arithmetical mean of nutritive value of individual proteins. Many proteins which are normally ingested have low nutritive values, but when taken in combination, a considerable advancement of the nutritive value of the whole is effected. However, it must be repeated, in order to effect a supplementation, two or more amino acid mixtures or proteins must be ingested simultaneously. The lapse of as short a time as an hour or two between ingestion of proteins that usually have mutual supplementation may result in complete failure in the synergy.

It has been possible on three different occasions for the writer to produce symptoms of milk fever in cows possessing the inherent predisposition towards the disease by the abrupt reduction of the protein ration during the last month or so of pregnancy. These animals were all on poor roughage and the concentrate allowance was not great before reduction. Within one week, animals exhibited weakness, developed edema of the fetlocks, which is generally characteristic of protein deficiencies in other species, and finally the syndrome referred to as the "knuckling type of milk fever." This is the type that usually proves refractory to calcium therapy. The cow ap-

pears fairly bright, usually eats, but cannot rise. Ankles double over when an attempt is made to get up. These cases and those met in the field respond best to protein therapy. Good quality protein can be supplied orally, or if prompt response is desired, it can be obtained by the daily infusion of a liter or more of a 15% solution of amino acids derived by the hydrolysis of casein (parenamine®). This is also the treatment of choice in calcium refractory cases.

Calcium-Protein Relationship

The part that protein plays in the milk fever complex does not belittle the role of calcium; rather it strengthens it and serves to explain failures in the use of calcium alone. In the blood, this mineral exists in two forms; ionized calcium and calcium as bound to protein. Normal values range from 9 to 12 mg./100 ml. blood, of which 4.5 to 6 mg. are diffusible, being the ionized and physiologically active form; while the remaining fraction is loosely bound to protein[13]. The amount of ionized calcium, from which milk calcium is drawn at onset of lactation, is dependent upon an equilibrium between total serum calcium and that portion bound to protein, the constant of the equilibrium being altered by such factors as pH and CO_2 tension. The ionized form is physiologically active in regulating neuromuscular irritability, and a decrease in its amount is followed by various degrees of muscular irritability leading to collapse[14].

It has been shown that the extent to which calcium is bound to protein is dependent upon the concentration of plasma proteins and it can be reduced in the presence of diminished plasma proteins[15]. Concentration of the plasma proteins is contingent upon protein balance of the animal. The degree of calcium dissociation has been shown to obey the mass law equation, at pH 7.35, provided the composition of the plasma protein be not altered greatly[16]. Madsen[17] has reported that a coexisting protein deficiency is often found in calcium and phosphorus deficient animals. This has been interpreted to mean that as long as the plasma protein concentration can be maintained it is possible for the cow to mobilize calcium from her skeletal reserves, which account for 90% of her total amount.

If this close relationship between calcium and protein is appreciated, it becomes imperative to supply a large quantity of the best quality of dietary proteins to the cow not only during lactation, but during the dry period, especially during the period of rapid development of the fetus. Pregnancy represents a normal condition during which there is a large demand for protein from the cow's body. This protein is transferred to the growing fetus, is present in the fluid surrounding the embryo, and is required for formation of the placenta and development of the udder. In addition, blood volume of the pregnant cow is increased, and this too requires additional protein. The greatest drain of this substance from the cow's body occurs at onset of lactation with secretion of colostrum. It thus becomes apparent that the present practice of feeding high carbohydrate feeds to milk fever susceptible cows during the dry period, without adequate allowances of protein of high biologic value, must be considered as a contributing factor to the disease. The tendency is towards adding weight to the animal, but the addition is chiefly adipose tissue. What is required is best explained in the words of a noted herdsman, "red meat."

Determination of Serum Proteins

In this area, the serum protein determinations of over 50 clinical cases of milk fever averaged 5.8 gm./100 ml. blood serum. Technic used was measurement of the specific gravity of the serum in solutions of copper sulfate[18,19]. Howe's[20] normal of 7.43 gm./100 ml. plasma has not been approached in cows with milk fever or ketosis, nor in heavy producers while milking or dry.

While serum protein determinations can be performed readily in the office by use of the copper sulfate specific gravity technic, they are subject to many influences and may be misleading. One has to consider factors of hemodilution and hemoconcentration, the latter of which rapidly develops in the comatose cow down with milk fever. Moreover, one has to realize that concentrations tell nothing about total circulating amounts of these complex substances, which in a variety of ways may become appreciably altered with little or no change in their concentrations. Thus, a simple laboratory test may not always give a reliable index of the true protein state of the cow. The most valid, although clinically unwieldy, method, of establishing an animal's protein nutritional state is by following its nitrogen balance after feeding large amounts of protein. The cow with a protein deficiency will retain large amounts of protein that the well nourished cow largely degrades to urea and am-

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monia. It, therefore, appears that the best way to determine which animals need more protein is by careful history-taking, with particular attention to previous attacks, feeding program, and production records; and by careful physical examination, looking for good muscular development. Keen clinical judgment is necessary to distinguish those in which change of weight is due mostly to deposit of fat. Patchy fat on the dairy cow long has been mistaken for good condition and it is in this type that incidence of milk fever and ketosis is the greatest.

Preventive Measures

Feeding experiments, initiated in 1946 and still in progress, using high quality proteins in the ration of cows having had either milk fever or ketosis have indicated that this procedure will reduce the number of recurrences and is useful in the therapy of both conditions. This feeding practice has been extended largely to all dry cows in herds cooperating in this study and the program has been carried out long enough to be evaluated. It was found that when roughage was good, increasing the amount of concentrate, 20% crude protein, up to 10 to 12 lb. daily in cows susceptible to either milk fever or ketosis was followed by sharp reduction in disease incidence. Where the roughage was poor, which is generally the case in this section, the increase in the grain ration did not materially change the incidence. This was contributed to lack of amino acids of high biologic value in the diet. Addition of animal proteins to the same ration prevented the development of the disease in a large percentage.

While the recommended procedure at present is the feeding of 1 lb. of dried skimmed milk daily for two or three weeks before calving and continuing for another week or ten days after, other sources of animal proteins have been fed with equally good results. These have included meat meal, animal liver meal, fish liver meal, glandular meal, and dried milk albumin. The problem of palatability of these supplements is the chief obstacle at the moment. Feeding the dried skimmed milk in beet or citrus pulp seems to be the most satisfactory way of getting the cow to consume it regularly. Animal proteins, by virtue of containing large amounts of essential amino acids, are superior to vegetable proteins in supplying raw material for protein synthesis, especially in the protein deficient animal. It also has been shown recently that animal proteins are rich in vitamin B_{12}, or the animal protein factor, with which it is now believed to be identical, and that this substance in small amounts increases the efficiency of the utilization of vegetable proteins. This would account for the marked increases in weights of young animals fed vitamin B_{12}.

There is no question that if knowledge of the amino acid composition and supplementation of vegetable protein mixtures was better understood, the same results might be obtained by their use. The addition of palatable calf feeds such as calf manna®, to regular dairy rations has helped reduce incidence.

McKittrick[21] has stated that in his purebred herd of Guernseys, there has been a significant reduction in the incidence of ketosis by the addition of 90 lb. of meat scrap to each ton of grain mixture. This amount would be sufficient to supplement vegetable proteins of lower nutritive values in addition to supplying a liberal amount of vitamin B_{12} to aid its utilization.

Results in our feeding experiments thus far have been sufficiently successful to have influenced two commercial concerns to modify fitting rations by the supplementation of animal proteins, and wide-scale use of these concentrates is now being given trial in this region.

It is the hope of this practitioner that this report, based chiefly on clinical observations, will serve to stimulate further investigation on the nutritional aspect of milk fever.

REFERENCES

1. Greig, J. R., Studies in the Prevention of Milk Fever. *Vet. Rec.*, 10:301, 1930.
2. *Idem*, The Nature of Milk Fever, 7th Intnat. Vet. Congress, London, 1930, vol. 3, p. 306.
3. Barker, J. R., Blood Plasma Changes and Variations in the Female Bovine Toxemias. *Vet. Rec.*, 51:575, 1939.
4. Udall, D. H., The Practice of Veterinary Medicine, ed. 5, Ithaca, 1947, p. 356.
5. Jones, L. M., Calcium and Udder Inflation in Milk Fever. *N. Am. Vet.*, 30:725, 1949.
6. Roberts, S. J., Recurrent Milk Fever. *N. Am. Vet.*, 31:109, 1950.
7. Bowen, L. E., Clinical Observations on Milk Fever. *Vet. Med.*, 38:154, 1943.
8. Eckles, C. E., Combs, W. B., and Macy, H., Milk and Milk Products, ed. 2, New York, McGraw Book Co., 1936, pp. 14-37 and 44-45.
9. Fearon, R. F., An Introduction to Biochemistry, ed. 2, St. Louis, C. V. Mosby Co., 1940, p. 129.
10. Tolly, S. J., *Bio. Rev.*, 15:421, 1940.
11. Borsook, H., Protein Turnover and Incorpora-

(References continued on page 238)

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Streptococcic Infections In Pigs

JOHN D. RAY, B.S., D.V.M., Omaha, Nebraska

STREPTOCOCCIC infections occur commonly in swine. In many instances they are not recognized. Animals which recover from clinical manifestations may continue to carry infection and become a menace to susceptible individuals. Pigs farrowed by sows so infected may develop navel lesions and pyemic arthritis. There is no practical method of identifying carrier animals until offspring exhibit evidence of infection. This is usually possible when pigs are from one to four weeks of age.

Until joints become distended, there is little on which to base a diagnosis. Pigs are depressed, with elevated temperature, ruffed hair coat, but move about and nurse abnormally. Animals only a few days of age which develop infections in the stifle and hock joints may refuse to bear weight on the hind legs and stand by bearing weight on the forefeet; balancing by placing the nose on the ground. Involved joints are hot, sensitive to touch, and fluctuate as a result of pus accumulation in the joint capsule. Respiratory complications are not uncommon. Organisms usually are those of the beta hemolytic group.

Pyemic Arthritis

Mortality as a result of pyemic arthritis is serious. Some recovered patients remain unthrifty for several weeks. Autopsy usually reveals pus in the region of the navel, sometimes extending to the bladder. Infected joints contain a characteristic creamy, yellow pus. Occasionally, pigs three weeks or more of age will show proliferations on the heart valves (so-called cauliflower growths) concurrent with navel and joint lesions.

Joint and heart lesions due to streptococci need to be differentiated from swine erysipelas. However, that disease is rare in such young pigs and the joint content in erysipelas arthritis is characterized by a flaky synovia. Cultures are necessary to determine the exact cause of the heart lesions if other findings are not diagnostic.

Pigs at weaning age or older may show induration of the joint region and on autopsy a mass of caseated pus can be demonstrated within the joint capsule. The mass takes the form of the space where located and may be firmly adhered to the fringe of tissue in the capsule. This lesion usually indicates that the animal was a victim of pyemic arthritis earlier in life and had overcome acute infection. Some old lesions are sterile, which is further evidence of recovery.

Courtesy Corn States Serum Company
Streptococcic arthritis

Control of streptococcic infections is important; otherwise, trouble may be anticipated from year to year. Sows which farrow pigs that develop pyemic arthritis early in life should not be kept for rebreeding, and infected pigs should be kept separate from healthy litters. Quarters should be thoroughly cleaned and disinfected. Antibiotics are indicated for treatment in acute stages of the disease. Proper housing and good nursing care increase the number of recoveries.

Pulmonary Edema

Respiratory complications are common and streptococci play a major role in many cases. These microorganisms may cause acute pulmonary edema, resulting in heavy death losses. A number of other infections produce similar lesions and it is necessary to differentiate etiological agents. Other common infections associated with acute edema of the lungs are Corynebacteria, *Erysipelothrix rhusiopathiae*, Salmonella and influenza (influenza virus associated with *Hemophilus suis*).

Symptoms of acute pulmonary edema are sometimes overlooked in warm weather as animals may show little more than a rapid

respiration for the first 24 to 36 hours. When observed during cold weather, lung lesions should be suspected immediately. A rise in temperature usually accompanies the increase in respiration. However, this may not occur if Corynebacteria are responsible agents.

As the edematous fluids gravitate in the lung, pendant parts fill up, collapsing air cells. When this occurs, the character of breathing changes to a jerky or thumpy type. The rate is slowed in proportion to the degree of filling. If resistance is high, edema begins to be resolved immediately. However, if certain destructive organisms become established, lesions may become chronic and terminate in suppuration and necrosis.

Treatment of the acutely sick has given encouraging results if started during the first 24 to 36 hours of onset. Procaine penicillin G (2,000 to 3,000 units per lb. of body weight) is recommended. Less favorable response is obtained when treatment is started after symptoms of thumpy breathing are evident. Combined sulfonamides have been reported as useful. Good bedding with properly ventilated housing facilities are important treatment adjuncts.

Abscesses

Abscesses in the throat region are common in some droves and cause farmers considerable anxiety. Streptococci frequently can be isolated from the pus. Whether these organisms are primary or secondary may not be discernible. Many of these cases, when examined carefully, show actinomycotic infections. In these, streptococci would be considered secondary. Iodine therapy, comparable to that used for actinomycosis in cattle, is indicated. Herd treatment markedly reduces the incidence of abscesses.

Otitis Media

Streptococcic infection in the inner ear of pigs is observed frequently. In some droves only one or two individuals are affected; while in others, a number of cases develop. Animals affected carry the head to one side and may exhibit incoordination. Meningitis and death may result from extension of infection. A careful autopsy is necessary to verify diagnosis. Treatment has been of no avail.

Streptococcic Enteritis

Streptococci have been associated with certain outbreaks of enteritis in swine. Their exact role has not been determined. Likewise, infection has been associated with gastritis and edema of the stomach. No doubt it is secondary in edema disease, based on present concepts.

Cellulitis

Cellulitis in swine appears as an acute local infection of the subcutaneous tissues of one or more legs. The legs swell and may become so tense that serum oozes through the skin. An

Acute cellulitis (streptococcic)

outbreak in a herd may be confined to a few animals or a large number may be affected.

A hemolytic streptococcus has been isolated in the cases examined. It is difficult to cultivate on artificial media from some outbreaks. This organism has been found to be highly pathogenic for mice or rabbits in a few tests made where edematous fluid was used as inoculum.

The infection becomes established on floors and bed boards. Any contusion of the skin favors an invasion and sand burrs have appeared to carry the infection into the tissues on occasion. Contaminated sales barn premises act as a reservoir. Incubation period is three to five days.

Prognosis varies. Considerable loss is experienced in some outbreaks, while in others, the recovery rate is good. If the lesion suppurates, the animal is likely to have a long convalescence.

Affected individuals should be isolated and contaminated premises thoroughly cleaned and disinfected. Removal of bed boards and other obstructing objects will aid materially in stopping the spread of infection. Experience with the use of antibiotics and sulfonamides in treatment has been too limited to justify a report.

(*Continued on page 241*)

Canine Coccidiosis: Diagnosis And Therapy With Coccithane

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FOR the past 25 years there has been a steadily increasing interest in investigational work on the problem of coccidiosis. Becker's[1] statement in 1934 that, "Coccidia probably cause a greater economic loss among animals of the temperate zone than any other group of protozoa" appears to be justified when consideration is given to the widespread incidence and yearly loss due to coccidiosis in the United States. This writer further states, "The realization of the economic loss traceable to Coccidia, which has resulted from the observations and studies of recent years, has led directly to a tremendous amount of interest in these microorganisms, with the result that scientific investigation of their activities and publication have been stimulated to the extent that the literature is becoming increasingly voluminous."

From these years of investigational work, we have been rewarded by increased knowledge of the many phases associated with this parasitic problem, *i.e.* identification of genera and species, host specificity, life cycle, etc., but our therapeutic knowledge has not kept pace with the discoveries of the protozoologist.

Incidence in Dogs

Briefly reviewed, the presence of Coccidia in the dog was observed almost a century ago by Virchow (1860), but he did not consider the bodies observed as parasitic organisms. Scattered reports were made by other observers during the latter half of the 19th century but it was not until the first quarter of the present century that work was earnestly begun and specific information regarding incidence in dogs was made known. Most writers, today, report the existence of the genera Isospora and Eimeria in the dog, the former being the most commonly found. Three species of Isospora are considered pathogens; *I. bigemina, I. rivolta* and *I. felis.* Most writers are in accord in that the genus Eimeria is less frequently observed and they report only the one species *E. canis* of pathological concern.

That canine coccidiosis is quite prevalent throughout this country, and especially in the South, is demonstrated as more and more investigational work is made public. Hutyra, Marek and Manniger[2] quote Flaherty as finding more than 50% of dogs infected in North America; Glassner[3] at Fort Collins, Colo., found 79% of 320 dogs examined during the summer of 1938, to be harboring coccidial sporocysts. Emmerson and Dashoff[4] in 1943 reported clinical coccidiosis as a common and highly fatal disease of dogs in greater Boston; Catcott[5] reported an incidence of 10.6 in 113 dogs examined at Ohio State University. Lee[6] found 13.8% of the dogs presented at Iowa State Clinic during the period of 1920 to 1932 to be infected with Coccidia. Whether the estimated percentage given by Flaherty is too liberal or the percentages given by the other writers are too low, remains to be established.

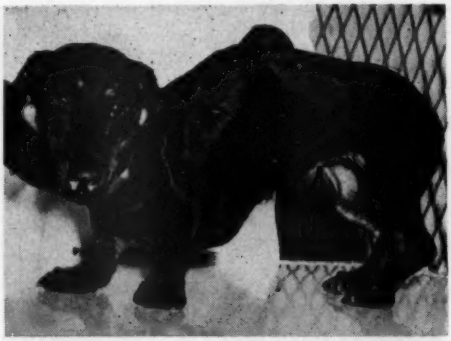

Fig. 1. Symptoms of coccidiosis frequently are confused with virus infection. Purulent nasal and ocular discharges are observed in some patients, accompanied by a slight cough. Extreme dehydration, such as exhibited by the Dachshund, may result from a persistent diarrhea.

The need of a unified, standardized procedure in conducting investigations with due consideration to seasonal variation is plainly apparent before accurate determination of the actual incidence can be made. Due to climatic conditions, infection in southern states is probably higher than in other areas. Wells[7] states that in his area (Florida) the number of clinical cases increased for successive seasons until

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it reached epizootic proportions in 1939. Efforts are being made at the Tuskegee Clinic to obtain seasonal and yearly incidence, using the most approved methods of fecal examinations.

Symptomatology

The mere observation of the presence of oocysts in fecal examination does not, of course, constitute the existence of clinical coccidiosis. However, the demonstration of large numbers of oocysts coincidental with manifestations of certain general and systematic symptoms and the recognition and elimi-

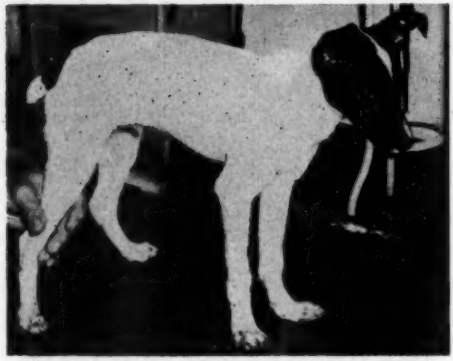

Fig. 2. Inappetence, listlessness, diarrhea admixed with blood and mucus, and a mild fever reaction of relatively short duration are regular observations in acute coccidiosis.

nation of any coexisting parasitism and infection will point the way to an accurate diagnosis. There is lacking in all available reports a clear-cut picture of the symptomatology, forms and course seen in clinical canine coccidiosis.

First, a review of the reports of other writers is most appropriate here in order to establish the fact that certain common definite symptoms have been observed by all writers though there is considerable variation in the occurrence or mention of other symptoms. Emmerson and Dashoff[4] report a diarrhea (usually no fresh blood) always present, often an elevated temperature, clamping of the teeth and frothing at the mouth. Locomotor incoordination was seen, generalized convulsions when observed indicated approaching death, and a few patients developed chorea. Puppies sometimes exhibited colic, bloating, grunting, pain on abdominal palpation, shrill howling and biting of hard objects. Anemia, emaciation, inappetence and emesis were also observed. Dyspnea from lung involvement, pustular skin eruptions and a purulent or mucopurulent conjunctivitis are also mentioned by these observers.

Hutyra, Marek and Manniger[5] consider coccidiosis as typically a disease of young animals and when seen in older animals no general effect is apparent as they remain healthy. These investigators state that the age incidence of severe infection is usually under six months, rarely up to 18 months. In the severe form, lassitude, reduced appetite, severe diarrhea with foul-smelling, slimy and sometimes hemorrhagic evacuations, progressive anemia and abdominal tenderness are seen. A milder form is described, passing undetected, terminating in recovery with little change observed in the intestinal evacuations. Wells[7] reports a prodromal symptom of dehydration present in the developmental period of about two weeks. This is not reported by others, though in the work at Tuskegee it was seen clinically or reported by clients in some cases.

Injected ocular membranes with possibly a purulent nasal discharge, fever varying in cases, mucus in formed or unformed feces or suddenly developing diarrhea are also symptoms mentioned by Wells[7]. Skin manifestations and nerve involvement were also observed and he calls attention to the close resemblance of the clinical picture of coccidiosis in many cases to that of distemper. Moss[8] mentions coccidiosis as a serious complication of canine distemper in Honolulu and believes that the course of clinical coccidiosis varies according to the type (based upon size) of oocysts present. His description is not complete, yet vomiting is mentioned as a serious symptom. McGee[9] considers the clinical manifestations to be most inconstant. He frequently observed fevers of 102.5 F. to 103 F. when cases were first presented; mucous or purulent ocular discharge; dry, sometimes sticky nostrils; gagging or hacking cough, and bowel movements varying from loose to bloody or mucus covered. In chronic cases, periodic diarrhea was observed or reported in the history, and occasionally posterior paralysis occurred. He further states that encephalitis or chorea was seen as a sequela in a few cases where no previous clinical evidence of distemper or other infectious disease existed.

Kirk[10] summarizes in this manner; "Coccidia induce sometimes acute but mostly chronic diarrhea. Young animals (two to six months of age) are most frequently affected though old ones are not exempt." Blood-stained bowel movements, depression, general weakness and anemia with gradual developing emaciation, complete the clinical picture

which might easily be confused with distemper. He mentions recovery of the oocysts in the vomitus and not in the feces, indicating localization of the disease in the gastric mucosa. The writer has not observed this nor found it mentioned in available literature. Chandler[12] records loss of appetite, emaciation, weakness, pallor, diarrhea with bloody feces and occasional fever. Morgan and Hawkins[18] describe bloody diarrhea, rapid progressive emaciation, palpation disclosing abdominal tenderness, anemia and general weakness as part of the clinical picture. They, too, observed muscular tremors of the posterior limbs and the possibility of confusing the condition with distemper.

Soon after the opening of the Small Animal Clinic at the Tuskegee Institute School of Veterinary Medicine in the fall of 1947, cases were received that presented the accepted picture of well developed cases of distemper. These were, for the most part, young dogs or puppies, and frequently an entire litter was affected. Ages usually ranged from two to four months. The constant history given in these cases was acute development with rapid loss of weight and inappetence. In some cases, a purulent nasal or ocular discharge was present as was a slight cough. In a small percentage of cases, the early stages of pneumonia were noted. Diarrhea with more or less blood and mucus either was existing at the time of entry or made its appearance 24 to 48 hours later. Marked anemia was constantly observed with varying degrees of emaciation. Attention was early directed to the absence of fever in most of these cases. Hookworms and roundworms often further confused the picture, as fecal examinations made on the day of entry, usually revealed some degree of infection with these parasites. Almost constantly present, however, were numerous oocysts. Where two or more puppies belonging to the same litter were presented, numerous oocysts were noted in the first fecal examinations from only one puppy but subsequent 24 hour examinations would reveal them in more or in all of the litter.

Following hookworm medication, no improvement occurred. Even where Ancylostoma and Ascaris infection were plainly negligible, the same course and end results were observed. Although distemper, either primary or coincidentally was considered at first, it was realized quickly that certain features were lacking, especially the absence of a diphasic fever. Fever was observed in some cases early in the course of the disease and lasted as a rule from one to two days. Symptomatic treatment was employed, but in most cases little was accomplished, the animals dying in from two to ten days.

Postmortem findings in these cases revealed a hemorrhagic enteritis present with extensive epithelial desquamation throughout the small intestines. More or less thickening of the mucosa existed. Further laboratory procedures confirmed the diagnosis of coccidiosis. This disease also frequently has presented a problem in the treatment of animals received affected with various other conditions, hindering progress and affecting recovery. In other words, a trauma case would be entered and the case progress favorably for two or more days, and then an enteritis, usually bloody, would develop. Fecal examinations had been made as usual upon entrance and any determined parasitism treated. Oocysts would not be present, but examination made at the appearance of enteritis would disclose numerous oocysts.

Since the recognition of the presence of clinical coccidiosis as a problem in this area, there has been ample opportunity over a three-year period to observe closely the variations of coccidial infection in dogs. There have been observed 106 cases of clinical coccidiosis during the period mentioned and it is the conclusion that the disease, at least in this area, manifests itself in three forms; namely, peracute, acute and chronic, with the acute being the most frequent. Apparently, degree of infection, age and other unrecognized factors influence the form.

Peracute Coccidiosis. In the peracute form, severe intestinal hemorrhage, alone or accompanied by epileptiform convulsions, appears early, giving little or no opportunity for treatment. Approximately 7% of the cases observed have been this form.

Acute Coccidiosis. The acute form may be considered as the prevailing or typical form and constituted about 90% of the total cases. The symptoms reported in the histories and observed in the clinic were in the following order: inappetence, listlessness, diarrhea with blood and mucus, and fever 102.5 F. to 103.5 F. of a one or two-day duration. The fever is present early in the course of the disease and seldom, if ever, recurs regardless of the complication or severity of the case. Fever may be missed entirely in those cases not presented early. Progressive emaciation and dehydration soon follow. Secondary symptoms, as purulent conjunctivitis, mild cough and slight purulent nasal discharge may appear

and in a few cases, pneumonic symptoms are common. Vomiting, when present, occurred early and presented a problem in treatment. Palpation of the abdomen is painful in practically all cases and acute dyspepsia is occasionally present. The course of this form is from two to three weeks, some time being required for the animal to regain a fair state of physical condition.

Chronic Coccidiosis. The chronic form was observed mainly in mature dogs and was evidently from continued reinfection without the acquisition of immunity or resistance. General unthriftiness and slow loss of flesh, persistent moderate anemia, continued or intermittent loose stools with more or less blood and mucus were observed. Fever was absent. The history given usually covered a period of weeks or months. Response to therapy in these cases is more rapid, as those observed were in good condition within two to three weeks following treatment. Table 1 shows the

TABLE 1. Clinical Manifestations Exhibited in 56 Cases of Clinical Coccidosis

Symptom	Number of Cases Observed
Fever	27
Anemia	56
Cough	13
Abdominal tenderness	56
Vomiting	16
Appetite	Poor to fair in all but one
Ocular and nasal discharge	10
Diarrhea	56
Convulsions	1
General condition	Poor to fair
Parasite identified*	
I. bigemina	37
I. rivolta	10
I. felis	12
E. canis	2

*Two or more were present in 5 cases.

frequency of the symptoms observed in the 56 cases considered in the evaluation.

The writer is completely in agreement with Wells[7] in not accepting the self-limiting idea so frequently mentioned in literature on coccidiosis. Canine coccidiosis as seen at our clinic is a highly fatal disease. Experiences as mentioned elsewhere in this article, particularly the high mortality before the use of coccithane,® attest to that fact. One must bear in mind in discussing coccidiosis the distinction between mere infection and clinical infection.

Cases selected and considered in this report are only those in which it was definitely established that coccidiosis was the primary factor acquired by natural infection and in those cases after the presence of other diseases was ruled out. Hookworms and distemper were the two principal complications considered. Where ancylostomiasis was seen to be a greater factor than coccidiosis or where distemper was known to be present, the cases were not considered. A follow-up of at least 95% of the cases has been possible.

Differential Diagnosis

In this area, hookworm infection is present to a great or less degree in about 99% of all clinical cases presented. The incidence is so great that the absence of hookworm ova in repeated fecal specimens examined from a specific animal excites comment. It, therefore, becomes necessary in those cases where both coccidia and hookworm infection jointly exist to decide which of the infections is the greater and is to be considered primary. The decision can be made by close observation of symptoms exhibited, together, with due regard to the results of fecal examination. There is no febrile stage present at any period in the course of ancylostomiasis, the very nature of the disease being conducive to normal or subnormal temperature. Ancylostomiasis, whether complicated or uncomplicated, is manifested by anemia which is more marked than in clinical coccidiosis and the fecal passages are more fluid and hemorrhagic with less mucus. A state of collapse ensues more rapidly, death often occurring within 24 hours after the recognition of illness in the animal by the owner. The response to therapy in ancylostomiasis cases received early enough to afford an opportunity for therapy, is rapid. In some instances recovery is phenomenal considering the great blood loss that has occurred. Unless complicated, the distemper-like symptoms seen in coccidiosis are absent.

Coccidiosis frequently is confused with distemper but may be differentiated easily if certain factors are kept in mind. It is an accepted belief that coccidiosis may serve as a primary factor just as does the virus of Carré in lowering general resistance and affording secondary organisms the opportunity to become pathogenic[14]. The appearance of these secondary symptoms associated with the

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presence of numerous oocysts should warn one of the possibility of coccidiosis. In the majority of clinical coccidiosis cases observed, fever is not diphasic, and in some of the cases included in this report a subnormal temperature was present for one or more days. Bearing in mind the diphasic fever characteristic of distemper and the time required for the development of the true distemper picture, observation of clinical coccidiosis will disclose that the distemper-like symptoms develop early, coincidental with fever. The period of acute illness is shorter though some cases may present as long a period of convalescence as is observed in viral infections. The most prevalent convalescence symptoms observed and reported have been a dry persistent cough or dyspepsia.

Treatment

Coccithane therapy was instigated after routine research procedures at the Pitman-Moore Research Farm indicated this drug reduced the number of oocysts present in stools following oral administration. Craige and Kleckner[15] published on the drug's taeniacidal properties in dogs. Blair and Burch[16,17] also reported on its effect on tapeworms in infected dogs and cats. Ryff[18,19] reported on its effectiveness in eliminating the fringed tapeworm, *Thysanosoma actinoides*, from the liver of infected sheep. A search of literature has not revealed any report on the use of this agent in protozoan infections in dogs.

The supplementary or symptomatic treatment employed in cases of clinical coccidiosis both before coccithane was used and after its evaluation began, has been identical. Where hookworms or ascarids were present, N-butyl chloride was given in an attempt to eliminate these parasites. Best results were obtained by giving small doses at 24 hour intervals until fecal examinations showed negative or nearly so. Results, especially since using coccithane, in attacking these parasites first, have been splendid in contrast to the experiences of McGee[9]. It has appeared logical and imperative that this be done first.

Normal canine serum, normal saline, alone or with dextrose, was given early to combat existing dehydration. Where the appetite was poor or entirely lacking, various protein hydrolysate compounds, mixed with canned milk, proved of value, as puppies often lap this mixture when all other food is refused. If refused, it was given forcibly for a day or two until voluntarily taken. If appetite was fair or active, rare meat mixed with protein hydrolysate compound was given for several days before offering animals the regular hospital diet. Vomiting, when present, often seriously interfered with the administration of both food and medication. In some cases, a definite gastric catarrh was present, the animals belching both food and drugs immediately or soon after administration. Anti-

Fig. 3. Fever is present early in the course of parasitic infection due to Coccidia and seldom, if ever, recurs.

nauseants, neutralizers and gastric sedatives were used in an attempt to control this symptom. Colloidal kaolin preparations were used to combat diarrhea. Where secondary infections appeared active, parenteral sulfonamides were used. Organic calcium and hematinic tonics have been valuable. Mixed canine distemper antiserum has been used routinely in all convalescent cases.

Mortality prior to using coccithane was high, being about 95% in those cases where secondary symptoms appeared and about 90% where only intestinal symptoms were present. Sulfamethazine did not give consistent or encouraging results though high dosages were maintained. The dietary precautions of McGee,[9] however, were not taken during its administration. Results obtained with sulfaguanidine did not differ from those obtained with sulfamethazine. Five of the acute cases (one litter) included in the report, table 2, had been under sulfaguanidine treatment before being presented at our clinic. One of the number was in epileptiform convulsions and all were heavily infected. In two of the chronic cases, repeated courses of sulfaguanidine had been given with no improvement. In a large

number of the cases, hookworm medication had been administered by the owner or a veterinarian prior to presentation at the clinic, thus eliminating that factor as substantiated by our fecal examinations. Several cases had been diagnosed elsewhere and the owner advised that no treatment was available.

Coccithane Administration

In most cases, coccithane was administered in daily doses of 2 gr. per lb. of body weight per day, divided into three doses. In a few cases the same total daily dosage was administered in two doses and recovery appeared to be delayed, apparently because presence of the drug in the small intestine could not be maintained. Increased peristaltic action, due to the diseased condition, is evidently present. Administration of 3 gr. per lb. of body weight divided into three doses produced no apparent undesirable results nor was the time of recovery materially lessened. In no instance did the drug produce emesis, regardless of the dosage or the time of administration. It was given preferably on an empty stomach or before feeding. In those cases where vomiting existed at the time of entry, coccithane did not influence the frequency nor induce emesis any more than did other medication or food. In all cases the drug was well tolerated. See table 2.

Comment

The writer's experience in recognizing clinical coccidiosis prior to coming to Tuskegee Institute may be considered negligible, as only in a few isolated cases had this entity been diagnosed in nearly 25 years of practice elsewhere. So forcibly and vividly have been presented the incidence and clinical phenomena of this disease in Alabama that one wonders, in retrospect, just how many cases have been overlooked or misdiagnosed in past years. It is the writer's belief that the incidence is greater generally than is commonly recognized, due to our too-ready acceptance of the "typical" distemper picture.

The writer feels that the evaluation presented may be open to criticism, particularly regarding the small number of controls used in the evaluation chart. As stated, 106 cases have been definitely diagnosed in our clinic and of these, 56 have received coccithane therapy. The remaining 50 not treated have served as controls. The mortality rates bear witness to the results with and without coccithane therapy. The primary interest after obtaining the drug has been to check the results obtained by its usage against results before its use.

It is recognized by the writer that there is need for much further work in investigating coccidiosis along combination therapy and possibly prophylaxis lines. Determination as to whether one species is more pathogenic than another is of importance. This had not been considered in the evaluation, yet the opinions of both McGee[6] and Wells[7] warrant consideration. Apparently both authors in mentioning small oocysts, referred to *I. bigemina* and called attention to their difficulty in dealing with this "type". Glassner's[8] statement that this species always matures fully in the subepithelial tissue of the villi may be the factor of importance. The concern in this evaluation has been to determine the efficiency of coccithane as a therapeutic agent without regard to genus or species and an investigation is being undertaken to determine species and genus efficiency which will, of course, require more cases and time. Also, the possibility of gastric mucosa and air passage involvement remains to be considered inasmuch as that belief has been expressed in literature.

Summary

1. Available literature relative to the incidence and symptomatology of clinical canine coccidiosis is reviewed.

2. The presence of a large number of oocysts in fecal examination, together with definite clinical symptoms and the elimination of all other primary factors, should warrant a diagnosis of clinical coccidiosis.

3. One hundred and six cases have been definitely diagnosed as clinical canine coccidiosis at the Tuskegee Institute Small Animal Clinic.

4. Three species of Isospora and one species of Eimeria genus were identified in the evaluation work. Both genera or two or more species may be present concurrently.

5. The disease manifested itself in three forms; peracute, acute and chronic.

6. Results in treatment had been poor and inconsistent using sulfamethazine or sulfaguanidine. Mortality varied between 90 and 95% depending upon the presence of secondary infection.

7. Fifty-six dogs were selected as typical definite cases of clinical coccidiosis to determine the efficiency and toxicity of coccithane in the treatment of this condition.

8. The dosage preferred was 2 gr. per lb. of body weight per day, divided into three equal doses.

TABLE 2. Clinical Evaluation of Coccithane in Cases of Canine Clinical Coccidiosis

Case No.	Breed	Age	Sex	Weight (lb.)	Dosage	Days Treatment Required for Negative Fecal	Total Days Treated	Results
1	Boxer	11 wk.	M	12	8 gr. t.i.d.	5	6	Recovered
2	Boxer	11 wk.	F	11	7½ gr. t.i.d.	7	8	Recovered
3	Boxer	11 wk.	M	11	7½ gr. t.i.d.	8	9	Recovered
4	Boxer	11 wk.	F	12	8 gr. t.i.d.	5	6	Recovered
5	Dachshund	12 wk.	F	6	Epileptiform convulsions (unable to administer drug)			Died 48 hr.
6	Dachshund	12 wk.	F	6	6 gr. t.i.d.	7	8	Recovered
7	Dachshund	12 wk.	M	6	6 gr. t.i.d.	6	7	Recovered
8	Dachshund	12 wk.	M	6	6 gr. t.i.d.	7	8	Recovered
9	Dachshund	12 wk.	F	6	6 gr. t.i.d.	8	9	Recovered
10	Foxhound	12 wk.	M	13	13 gr. t.i.d.	10	11	Recovered
11	Foxhound	12 wk.	M	10	10 gr. t.i.d.	10	11	Recovered
12	Foxhound	12 wk.	F	12	12 gr. t.i.d.	15	16	Recovered
13	Foxhound (control)	12 wk.	M	11	Pneumonia; sulfaguanidine, 1 gr. per lb. per day, divided into three doses			Died 7th day
14	Foxhound (control)	12 wk.	M	12	Diarrhea with severe hemorrhages; sulfaguanidine 1 gr. per lb. per day, divided into three doses			Died 18th day
15	Foxhound (control)	12 wk.	F	10	Diarrhea with severe hemorrhages; sulfaguanidine 1 gr. per. lb. per day, divided into three doses			Died 21st day
16	Foxhound (control)	12 wk.	M	13	Pneumonia; sulfaguanidine, ½ gr. per lb. per day, divided into three doses			Died 15th day
17	Collie	8 wk.		12	8 gr. t.i.d.	6	7	Recovered
18	Boxer	8 wk.		8	Peracute intestinal hemorrhages and vomiting, no medicine retained			Died 12 hr.
19	Boxer	8 wk.		7	Peracute intestinal hemorrhage and vomiting, no medicine retained			Died 24 hr.
20	Boxer	8 wk.		8	Peracute intestinal hemorrhages and vomiting, no medicine retained, nor food			Died 36 hr.
21	Boxer	8 wk.		7	4 gr. t.i.d.	6	7	Recovered
22	Boxer	8 wk.		8	5 gr. t.i.d.	7	8	Recovered
23	Boxer	8 wk.		8	5 gr. t.i.d.	5	6	Recovered
24	English setter	6 mo.		24	16 gr. t.i.d.	8	9	Recovered
25	Doberman	17 mo.		38	25 gr. t.i.d. (Had received 3 courses of sulfaguanidine at home)	5	6	Recovered
26	Great Dane	3 yr.		60	40 gr. t.i.d.	9	10	Recovered
27	Doberman	10 wk.		12	8 gr. t.i.d.	10	11	Recovered
28	Doberman	10 wk.		14	8 gr. t.i.d.	8	9	Recovered
29	Doberman	10 wk.		12	8 gr. t.i.d.	7	8	Recovered
30	Doberman	10 wk.		15	10 gr. t.i.d.	12	14	Recovered
31	Doberman	10 wk.		13	8 gr. t.i.d.	8	9	Recovered
32	Doberman	10 wk.		15	10 gr. t.i.d.	6	7	Recovered
33	Great Dane	3½ mo.		36	24 gr. t.i.d.	5	6	Recovered
34	Mongrel Shepherd	6 wk.		6	4 gr. t.i.d.	7	8	Recovered
35	Cocker Spaniel	4 mo.		12	8 gr. t.i.d.	6	7	Recovered
36	W.H.Fox Terrier	2½ yr.		18	12 gr. t.i.d.	7	7	Recovered
37*	Foxhound	11 wk.	F	12	12 gr. b.i.d.	Vomiting, persistent diarrhea, blood and mucus		Died 23rd day
38*	Foxhound	11 wk.	M	12	12 gr. b.i.d.	15	18	Recovered

TABLE 2 — *continued*

Case No.	Breed	Age	Sex	Weight (lb.)	Dosage	Days Treatment Required for Negative Fecal	Total Days Treated	Results
39*	Foxhound	11 wk.	F	11½	12 gr. b.i.d.	Vomiting, persistent diarrhea, blood and mucus		Died 21st day
40*	Foxhound	11 wk.	M	12	12 gr. b.i.d.	42	44	Recovered
41*	Foxhound	11 wk.	M	12	12 gr. b.i.d.	26	28	Recovered
42*	Foxhound	11 wk.	F	8	8 gr. b.i.d.	Vomiting, pneumonia developed on 6th day		Died 7th day
43*	Foxhound	11 wk.	F	10	10 gr. b.i.d. Pneumonia on 8th day	22	24	Recovered
44	German Shepherd	6 mo.	F	36	24 gr. t.i.d.	Pneumonia 3rd day. *I. felis, I. rivolta, E. canis*		Died 6th day
45	Cocker Spaniel	6 mo.	M	12	8 gr. t.i.d.	8	9	Recovered
46	Cocker Spaniel	4 mo.	M	9	6 gr. t.i.d.	9	10	Recovered
47	Shepherd	1 yr.	F	48	32 gr. t.i.d.	8	9	Recovered
48	Shepherd	2 yr.	M	60	40 gr. t.i.d.	10	11	Recovered
49	Pekinese	2½ mo.	M	6	4 gr. t.i.d.	8	9	Recovered
50	Pekinese	2½ mo.	M	3½	2 gr. t.i.d.	8	9	Recovered
51	Boxer	4 mo.	M	12	8 gr. t.i.d.	10	11	Recovered
52	Pointer	8 mo.	F	36	24 gr. t.i.d.	8	9	Recovered
53	Boxer	2 mo.	F	12	8 gr. t.i.d.	9	10	Recovered
54	Coonhound	3 mo.	M	12	8 gr. t.i.d.	12	12	Recovered
55	Coonhound	3 mo.	M	12	8 gr. t.i.d.	10	12	Recovered
56	Collie	3 mo.	M	24	16 gr. t.i.d.	11	12	Recovered

*Administration of drug twice daily appeared ineffective.

9. Results were satisfactory in all cases except where emesis was a pronounced symptom or where the total daily dosage was given in two doses. In no case was any evidence of toxicity shown. Mortality under this therapy was considerably under 10%.

10. The need of supportive treatment was recognized and was identical both prior to and after the employment of coccithane.

11. Clinical canine coccidiosis is not a self-limiting disease in many instances, the infection lowers resistance, secondary organisms become active, complicating and prolonging the course, with death resulting where treatment is inadequate.

Acknowledgments

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REFERENCES

1. Becker, E. R., Coccidia and Coccidiosis of Domesticated, Game and Laboratory Animals and Man. Ames, Iowa, Collegiate Press, Inc., 1934. Monograph no. 2, p. 1.

2. Hutyra, F., Marek, J., and Manninger, R., Special Pathology and Therapeutics of Domestic Animals, ed. 5, Chicago, Alexander Eger, Inc., 1949.

3. Glassner, F. X., Studies in Canine Coccidiosis. *Jour. Am. Vet. Med. Assn.*, 96:225, 1940.

4. Emmerson, F. H., Dashoff, A. D., Clinical Coccidiosis in Puppies. *Vet. Med.*, 38:32, 1943.

5. Catcott, Earl J., The Incidence of Intestinal Protozoa in the Dog. *Jour. Am. Vet. Med. Assn.*, 108:34, 1946.

6. Lee, C. D., The Pathology of Coccidiosis in the Dog. *Jour. Am. Vet. Med. Assn.*, 85:760, 1934.

7. Wells, John R., Canine Coccidiosis. *Jour. Am. Vet. Med. Assn.*, 47:240, 1939.

8. Moss, Lloyd C., Heartworm and other Parasitic Diseases of Dogs (Hawaiian Islands), *N. Am. Vet.*, 21:548, 1940.

9. McGee, H. L., Coccidiosis in the Dog, Clinical Observations. *Jour. Am. Vet. Med. Assn.*, 117:227, 1950.

10. Kirk, Hamilton, Index of Diagnosis, Baltimore, Md., Williams & Wilkins Co.. 1948, p. 155.

11. *Ibid.*, p. 160.

12. Chandler, Asa G., Introduction to Parasitology, ed. 8, New York, John Wiley & Sons, Inc., 1948, p. 225.

(*References continued on page 241*)

Attempts To Propagate The Virus Of Equine Infectious Anemia In Embryonated Eggs With Special Reference To Cultivation In The Yolk Sac

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THE extensive use, during the past decade, of developing chick embryos for the rapid propagation of various infectious agents has simplified greatly the procedure for maintaining the viability of such agents and has facilitated the study of different aspects of some infectious diseases.

Although chick-embryo inoculation technics have been employed to great advantage in studies of a number of virus diseases of animals and have resulted in the development of effective avianized vaccines against equine encephalomyelitis and rabies, they have been used to a limited extent only in studies of equine infectious anemia.

Pinus and Pirog[1] were apparently the first workers to attempt artificial cultivation of infectious anemia virus in chick embryos. These investigators employed the method of Goodpasture and coworkers[2] for exposure. They made numerous serial passages of the virus in fertile eggs, but no evidence was obtained that it had any lethal effect on the embryo either from primary injection or serial passage. Histological examination of 200 embryos exposed to the virus failed to reveal any alterations specific for infectious anemia. Two healthy foals injected subcutaneously with a suspension of pooled embryonic material from the tenth to 21st serial passages and blood of a 51-day-old chicken hatched from an egg injected with virus in the embryonic stage failed to develop any clinical symptoms of the disease after 90 days.

Hallauer and Moosbrugger[3] in 1949, also reported that attempts to transmit the virus to chick embryos was unsuccessful.

Early Investigation by the Bureau†

It appears appropriate here to review briefly some earlier attempts by Bureau investigators[4] to propagate the virus in embryonating eggs. The initial attempts were made as early as 1939. In these preliminary studies, approximately 0.2 cc. of filtered or unfiltered virulent blood serum from acute and chronic cases of infectious anemia was injected into the allantoic sac of fertile eggs incubated six to 11 days. Thirty serial passages at 24-hour intervals were made in eight-day embryonated eggs, and 37 serial passages at one to six day intervals in ten-day embryonated eggs. No definite evidence, however, was obtained that the primary injection or serial passage of the virus had caused any alteration in the macroscopic appearance or growth of the chick embryo. A few eggs from each lot injected with virus were allowed to hatch and these developed into apparently normal chickens.

Additional attempts were made at a later date to propagate the virus in chick embryos via the allantoic sac. Primary inoculation of seven- to 12-day embryonated eggs with six strains of virus failed to produce any significant alterations. Results were likewise negative when four to 19 serial passages were made with the six viruses in eight- and ten-day embryonated eggs. A few of the inoculated eggs from each group were allowed to hatch and produced apparently normal chicks.

During serial passage of the virus some embryos died, but in most instances sterility tests indicated that such deaths were due to bacterial contamination. Bacteriologically sterile, dead embryos, which are occasionally found in infected eggs, were likewise observed in uninfected controls incubated under identical conditions and were, therefore, considered of no significance.

To determine if multiplication of the virus occurred in the chick embryos after serial passage, a suspension of the embryo and embryonic fluids representing the terminus of the fifth serial passage of the New Hampshire strain of virus was injected into a normal horse, no. 1396. This animal remained well

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for 183 days, but when challenged with a virulent strain of infectious anemia virus developed acute symptoms of the disease after an incubation period of eight days. The results of this test indicated that the embryo suspension tested was non-virulent for horses and possessed no immunizing value against the disease (section A, table 1).

Intravenous Inoculation of Embryonated Eggs

During our preliminary studies an improved technic[3] was developed for both intravenous inoculation and bleeding of embryonated eggs. This was employed in all subsequent intravenous studies.

Thirty serial passages, designated as C series, with filtered virulent serum 1211 (California strain) were made intravenously at 24-hour intervals in 11-day chick embryos. Nine intravenous serial passages, designated as A series, were made at intervals of 48 hours in 11-day-old embryos with pooled virulent serum 1193 and 1194 representing a mixture of the Virginia, New York, and California strains of virus. Pooled virulent defibrinated blood of the same origin was similarly passed through a third group of eggs designated as B series. Primary intravenous injection of the virus or its serial passage in all three series failed to have any lethal effect on the chick embryo.

To determine if any multiplication of the virus occurred, the terminus of the 21st serial

TABLE 1. Results of Horse Inoculation Tests on Chick Embryo Suspensions for Virus or Immunizing Value

	Horse No.	Type of material (suspected of containing virus) injected	Results	Challenge: Days since original injection	Challenge: Strain of virus and amount injected	Challenge: Results
Section A	1396	15 cc. sq. of 5th serial passage N.H. virus via allantoic sac (suspension of chick embryo and fluids)	No reaction 183 days[1]	183	N.H. 50 cc. sq.	Reacted on 8th day; acute case[3]
	1348	Subinoculation of 50 cc. of blood from horse 1396 collected 72 days after receiving embryo suspension	No reaction 109 days[2]	109	Wyo. 50 cc. sq.	Reacted on 11th day; acute fatal case[3]
	1175	25 cc. sq. of 21st intravenous passage California virus (chick embryo suspension C series)	Mild reaction on 28th day. Temp. 101.8 F followed by 6 intermittent but mild reactions. No other symptoms. Questionable reactions	201	1st exposure N.Y. 50 cc. sq.	Mild reaction on 8th day[3]
				229	2nd exposure Va., Calif., N.Y. 850 cc. intravenously	Acute reaction with high temp. on 7th day[3]
Section B	972	Subinoculation of 50 cc. of blood from horse 1175 collected 201 days after receiving embryo suspension	No reaction 103 days[2]	103	N.Y. 50 cc. sq.	Reacted on 21st day; subacute case[3]
	1149	Same except 100 cc. of blood from horse 1175 collected 201 days after receiving embryo suspension	No reaction 75 days[2]	75	N.Y. 50 cc. sq.	Reacted on 34th day; subacute case[3]
	1224	40 cc. sq. 6th and 7th passage (A-B series) 25 cc. sq. 9th passage (B series) (embryo suspensions from intravenous passage)	No reaction 132 days[1]	132	N.Y. 100 cc. sq.	Reacted on 15th day; subacute case[3]
	1207	Subinoculation of 50 cc. of blood from horse 1224 collected 132 days after receiving embryo suspensions.	No reaction 161 days[2]	161	N.Y. 25 cc. sq.	Reacted on 25th day; subacute case[3]

[1]Indicates chick embryo suspension was non-virulent for horses.
[2]Indicates blood from horses injected with embryo suspensions was non-virulent.
[3]Indicates that horse was susceptible and material first injected had no immunizing value against the virus of infectious anemia.

passage of the C series was injected into normal horse 1175. On the 28th day after exposure, this horse developed a mild febrile reaction followed by six additional mild temperature reactions but with no other clinical symptoms. This was considered as a questionable reaction since blood samples collected 201 days after exposure were found to be avirulent when injected into two normal horses, 972 and 1149. However, all three horses when subsequently exposed to a known active strain of virus developed infectious anemia (section B, table 1). The embryonic suspension from the 21st serial passage was also injected into mice intracranially, intraperitoneally, and subcutaneously. All injected mice remained well for 60 days. The pooled embryo suspension of the sixth, seventh, and ninth intravenous serial passages (A and B series) was injected into test horse 1224, which remained normal for 132 days. Blood samples collected from this animal on the 132nd day after exposure, when injected into normal horse 1207, failed to produce symptoms of infectious anemia. On the other hand when both these horses were exposed to the New York strain of virus they promptly developed clinical symptoms of the disease (section B, table 1). These tests indicated that the embryo suspensions were non-virulent for horses and induced no immunity against the disease following its injection.

Attempts to Cultivate the Virus in the Yolk Sac of Embryonated Eggs

The advantages of the yolk sac method of inoculation over other methods of chick embryo inoculations for the propagation of infectious agents have been pointed out by Cox,[6] McNutt,[7] and Shope, *et al*[8]. Since the attempts to propagate the virus in the developing chick embryo via the allantoic sac and intravenously were unsuccessful, it was deemed advisable to ascertain if the yolk sac route of injection would be a satisfactory medium for growing the virus.

For primary exposure 0.2 cc. of virulent filtered serum from horses experimentally infected with infectious anemia was injected into the yolk sac by the method of Cox[6] and McNutt[7]. The strains and source of virus used in primary passages are shown in table 2.

In our studies on serial passage of the virus, three separate sets of egg cultures were carried in series, one set using the fluids; another, the embryo; and the third, the yolk material from six-day embryonated eggs that had been inoculated originally via the yolk sac with 0.2 cc. of filtered virus (New Hampshire strain). Successive serial passages were thus continued in eggs for 21 consecutive times at intervals of four days, all inoculations being made via the yolk sac. For inoculation purposes, suspensions of the yolk sac material and the chick embryos which had been triturated in a Ten Broeck grinder were diluted with two to three parts of sterile physiologic saline. Tests were made for bacteriologic sterility of all suspensions used for inoculating embryonated eggs.

TABLE 2. Strains and Source of Virus Used in Primary Passages of Infectious Anemia Virus via the Yolk Sac in Embryonated Eggs

Strain of virus and source	Type of virulent material injected
Calif.—1353 carrier	Filtered serum lyophilized
N.Y.—1420 acute	"
Ky.—1406 subacute	"
Wyo.—1295 carrier	"
Wyo.—1459 acute	Filtered serum
Neb.—1477 acute	"
N.H.—1461E acute	"
N.H.—1449 acute	"
Pooled strains N.Y.—1422 and Wyo.—1459 acute	"
Pooled strains Neb.—1380 N.H.—1449 Acute	"
None	controls

Following inoculation each lot of injected eggs, together with a number of non-inoculated eggs of the same age used as controls, were placed in the incubator in a horizontal position with air sac uppermost. The position of the eggs was changed once or twice daily. A temperature of 99 to 100 F. was automatically maintained with provisions for adequate humidity. All the inoculated eggs in each group, as well as the controls, were candled daily for the detection of dead embryos.

Horse inoculation tests were carried out to determine if any multiplication of the virus occurred from serial passage in the chick embryo. For this purpose, healthy susceptible horses were injected subcutaneously with suspensions prepared from the embryo and embryonic fluids, from different serial passages.

The results showed that primary inoculation of 0.2 cc., via the yolk sac, of ten samples of virulent filtered serum into six-day embryonated eggs (table 2) failed to have any lethal effect on the embryos. All remained alive during incubation, eventually hatched and produced chicks that appeared normal. Likewise no evidence was obtained that the

virus could be cultivated in six-day embryonated eggs by serial passage, at four-day intervals via the yolk sac, of egg fluids, embryonic material or egg yolk material.

Although a few died from bacterial contamination during serial passage, the virus appeared to have no lethal effect on them.

On macroscopic examination, lesions pathognomonic for infectious anemia were not observed in the embryos of injected eggs.

Some inoculated eggs in each series were allowed to incubate to the 21st day. Virtually all of them hatched and produced apparently normal chicks.

Susceptible horses inoculated with suspensions of embryos and embryonic fluids from the fourth, fifth, and sixth, or the 15th serial passage failed to develop symptoms of infectious anemia. However, all the test horses developed the disease when subsequently exposed to a virulent strain of New Hampshire virus, indicating that the chick embryo suspensions previously administered had imparted no immunity against the infection (table 3).

Filtered serum obtained from the inoculated horses 20 to 60 days after the administration of embryonic suspensions was injected into the yolk sac of six- or seven-day embryonated eggs. It produced no ill effects on the embryos as all the eggs injected with this material hatched and produced apparently normal chicks.

Summary

Unsuccessful early attempts to propagate the virus of equine infectious anemia in embryonated hen's eggs via the allantoic sac and intravenously are briefly reviewed.

In the present experiments, attempts to propagate the virus in embryonated eggs by primary injection or serial passage via the yolk sac were also unsuccessful. Neither primary injection nor serial passage of the virus produced any alterations in the macroscopic appearance of the embryo or any lethal effect on the embryo. Susceptible horses injected with suspensions of embryos and embryonic fluids from different serial passages of the virus failed to develop clinical symptoms of infectious anemia but when exposed to known virus promptly developed symptoms of the disease, indicating that the embryonic suspensions were avirulent and devoid of immunizing value for horses.

REFERENCES

1. Pinus, A. A., and Pirog, P. P., Versuch der Zuchtung des Virus der ansteckenden Blutarmut auf

(*References continued on page 234*)

TABLE 3. Results of Horse Inoculation Tests on Chick Embryo Suspensions for Virus or Immunizing Value

Horse No.	Material Injected	Amount and method	Results	Challenge		
				Days since original injection	Strain of virus and amount injected	Results
1464	Chick embryo suspension terminus from 4th yolk sac passage of N.H. virus 1461	20 cc. sq.	No reaction 60 days[1]	60	N.H. 10 cc. sq.	Reacted on 14th day; acute fatal case[2]
1462	Same as above but terminus from pooled 5th and 6th passage	20 cc. sq.	No reaction 74 days[1]	74	N.H. 20 cc. sq.	Reacted on 13th day; subacute case[2]
1426	Same as above but terminus from 15th passage	20 cc. sq.	No reaction 74 days[1]	74	N.H. 20 cc. sq.	Reacted on 13th day; subacute case[2]
1400	Chicken embryo suspension from normal 7-day incubated egg. (Control)	20 cc. sq.	No reaction 60 days[1]	60	N.H. 10 cc. sq.	Reacted on 15th day; subacute case[2]
1485	Filtered virulent serum 1461 N.H. strain diluted 1 to 2. (A control on virulency of virus injected in yolk sac of embryonating eggs)	10 cc. sq.	Reaction on 17th day (acute case)[3]	None		

[1]Indicates embryo suspension was non-virulent for horses.

[2]Indicates horse was susceptible to New Hampshire strain of virus and chick embryo suspension failed to immunize.

[3]Indicates virus used in chick embryo inoculations was fully virulent.

Streptomycin In The Treatment Of Streptococcic Mastitis†

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THE spectrum of antibacterial activity of streptomycin appears quite broad. Although it[8] has been used largely against Gram-negative organisms, *in vitro* trials indicate that it has considerable activity against the Gram-positive organisms *Streptococcus agalactiae, Staphylococcus aureus,* and *Corynebacterium pyogenes.*

Schalm[1] demonstrated the effectiveness of streptomycin (calcium chloride complex) in treatment of a limited number of cases of coliform mastitis. Benson[2] reported cure of mastitis due to *Escherichia coli* in a single quarter with two infusions of 0.5 gm. of streptomycin dissolved in 80 cc. of sterile distilled water with an interval of 12 hours between infusions.

Lipman[5] also reported success with streptomycin therapy in *E. coli* mastitis; streptomycin apparently did not have therapeutic value for staphylococcic mastitis.

The experiments of Foley and Byrne[4] indicated that combinations of streptomycin and penicillin are effective against mastitis caused by *Str. agalactiae* alone or when a concurrent infection with *Staph. aureus* also existed. All the infected quarters in their trial were treated with the streptomycin and penicillin combination and it is, therefore, impossible to evaluate the results accurately. The efficacy of penicillin in streptococcic mastitis has been established[7,9].

Cooper, Ford and Robinson[6] concluded that a mixture of streptomycin and penicillin effected a high percentage of cures in mastitis caused by a mixture of streptococci, micrococci and coliforms.

The study here reported was undertaken in an effort to determine the therapeutic value of streptomycin in mastitis caused by *Str. agalactiae.*

Material and Methods

All the experimental herds were on farms in the immediate area of Madison, Wis. Herds were maintained under average dairy farm conditions and had numerous cases of mastitis. Each group was tested by individual quarter sampling, utilizing the Hotis test, direct microscopic films of incubated samples and streaking each incubated sample upon a blood-agar medium containing 1:10,000 sodium azide and 0.5% mannitol. The infected quarters were divided on a herd basis into two nearly equal and comparable groups so that each medicament was employed under similar conditions. A single infusion of a drug was employed for each affected quarter in a diluent of 10 ml. of penicle®. Following treatment, each infected quarter was sampled weekly for three consecutive weeks by means of the same procedures which were employed in determining the original infectivity. A quarter was considered free of streptococci if it was negative to all the consecutive post-treatment tests during the three-week period.

Experiment 1. Streptomycin (calcium chloride complex**) compared with crystalline penicillin.

Group A—100,000 units of amorphous penicillin** sodium in 10 ml. of penicle (water-in-oil emulsion type vehicle).

Group B—0.5 gm. of streptomycin (calcium chloride complex) in 10 ml. of penicle.

The results of experiment 1 were as follows:

Group	Number Quarters Treated	Quarters Freed of Infection Number	%
A	87	33	37.9
B	96	26	29.2

The statistical evaluation of the results of therapy in experiment 1 revealed that 0.5 gm. streptomycin (calcium chloride complex) was as effective as 100,000 units of amorphous penicillin sodium, in the procedures utilized in this study.

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®Wallace Laboratories, Inc., New York, N. Y.

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Experiment 2. Dihydrostreptomycin** compared with crystalline penicillin.

Group A—100,000 units of crystalline penicillin G, potassium salt in 10 ml. of penicle.

Group B—0.5 gm. of dihydrostreptomycin in 10 ml. of penicle.

The results of experiment 2 were as follows:

Group	Number Quarters Treated	Quarters Freed of Infection Number	%
A	77	46	59.7
B	82	45	54.9

The statistical evaluation of experiment 2 indicates that 0.5 gm. of dihydrostreptomycin in 10 ml. of penicle is as effective a therapeutic agent for mastitis caused by *Str. agalactiae* as 100,000 units of crystalline penicillin G potassium salt, in 10 ml. of penicle, water-in-oil emulsion. (The chi-square value is 0.3835 which indicates there is no evidence of a difference in the effects of the treatments utilized.)

Discussion

The unsatisfactory results obtained in experiment 1 probably may be ascribed to the inferior sanitary conditions which prevail generally during the summer harvest season. The results of this experiment are not impressive since in other trials the percentage of recoveries among control animals given sterile saline or mineral oil, has been similar to that obtained in experiment 1 of this study.

In experiment 2, dihydrostreptomycin was as effective as penicillin in therapy of cases of mastitis associated with *Str. agalactiae*. Such activity is especially significant since the antibacterial activity of dihydrostreptomycin also encompasses *E. coli* infection[3].

Summary

1. Streptomycin (calcium chloride complex) 0.5 gm. in 10 ml. of penicle, was as effective in comparative tests, as amorphous penicillin sodium, 100,000 units, in penicle in treating mastitis caused by *Str. agalactiae*.

2. Dihydrostreptomycin, 0.5 gm., in 10 ml. of penicle was as effective as 100,000 units crystalline penicillin G, potassium salt, in the same vehicle in therapy of mastitis caused by *Str. agalactiae*.

REFERENCES

1. Schalm, O. W., Preliminary Observations on the Use of Streptomycin for the Treatment of Mastitis caused by Gram-negative Bacteria. *Cornell Vet.*, 38:186 (April), 1948.

2. Benson, D. V., Treatment of Bovine Mastitis with Tyrothricin, Bacitracin, Streptomycin, and Penicillin. *Jour. Am. Vet. Med. Assn.*, 111:289 (Oct.), 1947.

3. Lipman, Arthur, Report on Streptomycin in Mastitis. *Jour. Am. Vet. Med. Assn.*, 112:377 (May), 1948.

4. Foley, E. J., and Byrne, J. V., Observations on the Treatment of *Streptococcus agalactiae* and *Staphylococcus aureus* Udder Infections with Mixtures of Streptomycin and Penicillin. *Cornell Vet.*, 39:243 (July), 1949.

5. Cooper. G. W., Ford, C. M., and Robinson, G. R., Some Sulfa Drugs with Penicillin, and Dihydrostreptomycin with Penicillin, as Infusions in the Treatment of Mastitis. *N. Am. Vet.*, 31:656 (Oct.), 1950.

6. Waksman, Selman A., Streptomycin, Its Nature and Practical Application, Baltimore, The Williams & Wilkins Company. 1949, p. 91.

7. Spencer, G. R., Kraft, M. E., and Underbjerg, G. K. L., Efficacy of Intramammary Infusion with Penicillin and Diluents in Streptococcic Mastitis. *Am. Jour. Vet. Res.*, 8:325, 1947.

8. Kraft, M. E., and Spencer, G. R., *In vitro* Effect of Certain Antibacterial Agents on Organisms Encountered in Bovine Mastitis. *Proc. Soc. Exp. Biol. Med.*, 70:176, 1949.

9. Stults, A. W., and Foley, E. J., Treatment of *Streptococcus agalactiae* Mastitis by Single Injections of Penicillin in Retaining Vehicles. *Jour. Am. Vet. Med. Assn.*, 113:68 (July), 1948.

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Hühnerembrionen. *Sowyetskaja Veterinaria*, No. 8, 1937. Translation by Liaison and Protocol Section, OMGUS, G-V-1, p. 10.

2. Hallauer, C., and Moosbrugger, G. A., Research on the Virus of Equine Infectious Anemia. *Schweiz. Arch. Tierheilk*, 91:28 (Nov. Spec. No.), 1949.

3. Goodpasture, E. W., and Anderson, Katherine, The Problem of Infection as Presented by Bacterial Invasion of the Chorioallantoic Membrane of Chick Embryos. *Am. Jour. Path.*, 13:149, 1937.

4. Reports of the Chief of the Bureau of Animal Industry 1939, 1940, 1941 and 1949.

5. Eichhorn, E. A., A Technique for the Intravenous Inoculation of Chick Embryos. *Science*, 92:245 (Sept. 13), 1940.

6. Cox, Harold R., The Use of the Yolk Sac of Developing Chick Embryos as a Medium for Growing Rickettsia of Rocky Mountain Spotted Fever and the Typhus Group. *U. S. Pub. Hlth. Repts.*, 53:2241, 1938.

7. McNutt, S. H., A Partial Comparative Estimate of Three Methods for the Propagation of Infectious Agents in Developing Chick Embryos. *Vet. Stud.*, 4:9, 1943.

8. Shope, Richard E., Maurer, Fred D., Jenkins, Dubois L., Griffiths, Henry J., and Baker, James A., Rinderpest: IV—Infection of the Embryos and Fluids of Developing Hens' Eggs. *Am. Jour. Vet. Res.*, 7:152, 1946.

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Report Of A Series Of Deliberate Warfarin Poisonings: Suggested Treatment

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THE use of warfarin, 3-(alpha phenyl-beta acetyl ethyl)-4-hydroxycoumarin) in the extermination of mice, rats, and other pests of the rodent family has not been regarded with alarm by veterinarians, except where this preparation has been used indiscriminately.

While there have been reports of deaths of household pets following ingestion of this compound, these incidents are comparatively few in relation to its growing popularity for ridding premises of rodents. This may be due to the fact that when warfarin is employed in the proper amounts, there is comparatively little danger to pets. Yet, this is a problem of the veterinarian once a dog has been poisoned, since the animal is usually presented in a serious, if not moribund, condition.

The present series of 61 cases was one in which poisonings were deliberate. They resulted from the attempt by a mentally unbalanced individual, with an intense hatred for dogs, to rid the neighborhood of them. Large, even massive, doses were used to coat meat and bones. Poisoned food was then placed in the many vacant lots in the vicinity. The poisoner was clever in selecting a new compound and one for which an effective antidote had not been found.

The writers' attention was brought to this mass slaughter of dogs on July 28, 1951, by the death of the pet owned by one of us (H. W. B.). This was regarded as an isolated case until it was learned that six other dogs in the neighborhood had died of poisoning on July 28 and 29.

Naturally, curiosity was aroused and investigation revealed that purchases of warfarin in large quantity had been made by an individual, from a number of pharmacies in the locality; never more than two or three cans in one store, but a total of 39 cans. There may have been additional purchases since inquiry was confined to a half-mile radius. Further investigation revealed that the poisoner lived in a modern apartment house, quite new, and that it was entirely free of mice and rats. In addition, it was learned that she had threatened to "kill all the dogs in the neighborhood."

Proof of the nature of the poison was obtained when searching parties, made up of dog-owners, found meat and bones liberally dosed with a gelatinous-appearing substance which was fed to test animals with results which will be given. All dog-owners in the area were warned to bring in their pets at the first sign of illness.

It was noted that smaller dogs were more acutely ill than the larger ones and that there were more deaths among them. The following table will demonstrate this:

	Cases Reported	Deaths
Dogs under 15 lb.	33	10
Dogs 16 to 25 lb.	16	3
Dogs 25 lb. and over	12	1
Total	61	14

The fact that there were fewer reported cases among larger dogs can be explained on the basis of toxicity per unit weight and, further, that the owners of larger dogs exercised their animals secured by a leash.

The owner of the warfarin patent[1] has issued a bulletin on the toxicity of this compound in which is stated that 5 mg. per kg. of body weight, administered daily, would be fatal to dogs in from five to 15 days. Judging from the amount of poison recovered from the bait, each dog in our series must have ingested at least 1 to 3 gm. of the compound at one time.

Pathological Findings

Ten of the 14 dogs killed by the poison came to autopsy.

General findings were negative except for severe petechial hemorrhages of the intestinal mucosa. In two of the dogs, hemorrhages of the lungs were observed. There were no ulcerations of any kind in the stomach or digestive tracts as would have been the case if caustic poisons, such as phosphorus or sulfur, had been ingested. There was no paralysis nor convulsions that suggested poisoning by strychnine. None of the 61 dogs in this series exhibited neuromuscular symptoms.

In all cases, a small amount of heart blood was collected for test, but proved negative for known poisons, as did samples of stomach contents.

Laboratory Findings

In some patients which recovered, gastric lavage was performed and contents recovered were mixed with meal and fed to rats. None of these test animals died, indicating that the poison had been ingested by the dogs hours prior to the onset of illness and had been absorbed from the stomach.

The gelatinous material found on the poisoned meat and bones gathered in the vacant lots, when fed to rats and mice, killed all of them within 12 to 72 hours; all revealed the same symptoms as the dogs, *i.e.*, hemorrhage, shock, collapse, and death. Pathological findings on autopsy also were similar.

It may be deduced, therefore, that the poison found on the bait killed both dogs and rats and that this compound raised the coagulation time of the blood to such a point that seepage through the minor vascular vessels occurred.

To verify results, several cans of warfarin were purchased and the contents fed to ten laboratory rats. Rats 1, 2, 3, and 4 were fed the dosage recommended in the bulletin[1] on a mg./kg. weight basis (0.4 mg. per kg. of body weight). Rats 5 and 6 were given 0.8 mg. per kg. of body weight, and rats 7, 8, 9, and 10 were administered 2 mg. per kg. of body weight, which was considered to be the minimum quantity ingested by the dogs in our series.

Five mice were next given 2 mg. per kg. body weight and all these animals died within 24 hours after administration. Autopsy findings were the same as those in dogs and rats. Only a chemical of the coumarin or dicoumarin groups could produce findings of this nature.

Treatment of Canine Patients

On admission, 2 cc. of koagamin®[1] were administered intravenously to the dogs. Since most of these animals were in shock and collapse, this was followed by 200 to 300 cc. of glucose and saline solution into which 2 cc. of koagamin were incorporated. Vitamin K, 2 cc. (2 mg.), was injected intramuscularly. In some cases, respiration was so shallow that metrazol,®[2] 1 cc., was administered. If necessary, 1 cc. of koagamin was injected half-hourly or hourly until the animal was out of danger.

Results of Test Feeding

Rat No.	Dosage	Days Administered	Result
1	0.4 mg.	4	Died
2	0.4 "	5	Saved
3	0.4 "	5	Died
4	0.4 "	5	Died
5	0.8 "	3	Died
6	0.8 "	2	Died
7	2.0 "	1	Died
8	2.0 "	1 feeding	Died
9	2.0 "	2 feedings	Died
10	2.0 "	1 feeding	Died

In 16 of the first 30 cases saved, gastric lavage was performed but, apparently, this was not indicated since no poison could be found in the gastric contents.

Whether the 47 dogs whose lives were saved survived because of one or all of the therapeutic measures employed was not determined, but after the poison agent was identified, only seven of 54 animals died; this in contrast to the first seven dogs that were lost because of the obscure nature of symptoms. It might be said that most of these animals would have recovered spontaneously, but this is doubtful since they presented a picture of complete collapse. Naturally, routine treatment on every animal was carried out. An effort was made to save the lives of as many dogs as possible and not to sacrifice a single one to the interest of the method. It was an effort of the clinic, not of the laboratory.

However, since vitamin K takes no less than three hours to become effective in dogs, it is believed that koagamin and koagamin plus hypodermoclysis sustained these animals until vitamin K restored normal coagulation time.

The choice of koagamin was based upon the fact that one of the authors (H. W. B.) had had considerable experience with this preparation in human medicine over a period of years. The coauthor (B. S. K.)[3] described a series of cases in which hemorrhage was a factor and in which bleeding had been controlled by its use. Blamey,[3] in a clinical study, tested the value of koagamin in chemical poisoning and was enthusiastic over the results he obtained.

There is no evidence that koagamin affects prothrombin time. It is an emergency drug which acts directly upon the clotting mechanism and which controls seeping hemorrhage. It is non-toxic and repeated doses may be given at half-hourly intervals without trepida-

®[1]Chatham Pharmaceuticals, Inc., Newark, N. J.
®[2]Bilhuber-Knoll Corp., Orange, N. J.

tion. Since the action of warfarin, without doubt, raises the prothrombin time of animals, vitamin K is indicated. However, as stated, the initial action of this agent does not manifest itself for several hours after administration. Therefore, a coagulant such as koagamin is indicated to hold the fort, as it were, until the prothrombin time can be restored. The initial dose of 2 cc., intravenously, was decidedly an emergency measure and since, in most cases, the dogs demonstrated shock due to blood loss, treatment was supplemented with 200 to 300 cc. saline-glucose solution to which 2 or 3 cc. of koagamin had been added. If necessary, hourly injections of 1 to 2 cc. of koagamin were administered.

Usually, by the second day, patients were so far recovered that milk or milk and pablum was offered, to which a few drops of brandy had been added. Sulfasuxadine (about 2 gm.) was given routinely in all cases to prevent infection.

The first seven deaths were unavoidable since the nature of the poison was not known. Thereafter, 54 additional poisoning cases were brought to the writers' attention and only seven of these failed to recover. These animals were *in extremis* and therapeutic measures failed.

Discussion

The writers have not prepared this article with the intention of condemning the use of warfarin in the extermination of rodent pests. The malicious poisoner concerned in this series of events, an extremely well-read and cultured person, had an overpowering hatred for dogs. Any other poison might have been employed, *i.e.*, arsenicals, strychnine, mercury, etc.

Identification of the poison was based upon the following:

1. Symptoms of shock and hemorrhage observed.

2. Duplication of symptoms when laboratory animals were fed the poisoned bait.

3. Reproduction of symptoms when rats and mice were fed warfarin directly from sales containers.

4. Unusually heavy purchases of warfarin by a person who had no legitimate reason to buy such quantities.

5. The threat of the poisoner to get rid of all dogs.

It is unusual to dedicate a report such as this, but the writers trust that, in this case, permission be granted to depart from custom and dedicate this article to Brownie, the little dog whose death was responsible for opening up this line of investigation and because of whom so many other dogs have lived.

Summary

1. A series of 61 cases of deliberate warfarin poisoning of dogs has been described. The first seven dogs died because of failure to correlate the cases or to identify the poison. After the poison was identified, 47 of the remaining 54 dogs which were treated were saved.

2. All dogs presented hemorrhage in some form, either in bloody vomitus and stools or in a final fulminating oral hemorrhage immediately before death. In addition, all animals showed cyanosed membranes, rapid pulse, shallow respiration, shock and subnormal temperatures.

3. Laboratory tests on rats and mice, using the poison found on the bait, proved to be warfarin when tested against the commercial product. The laboratory animals showed the same symptoms of poisoning as did the dogs.

4. Treatment with koagamin, 2 cc. intravenously or 2 cc. in 200 to 300 cc. glucose-saline solution intravenously and vitamin K intramuscularly, was successful in saving 47 animals. Routine measures were employed thereafter. Three dogs in this series died when koagamin was not available.

Typical Case Histories

Case 1. Brownie, female, eight years of age, weight 10 lb. miniature Fox Terrier. At 7 a.m. on July 28, the animal awakened her owner (H. W. B.). She was whining and in evident pain. There were six pools of gray vomitus, as well as three formed stools found at various points. There was no sign of hemorrhage. The animal's respirations were rapid, shallow (gasping); temperature was subnormal. At 9 a.m. the animal was taken to the hospital. After examination, a tentative diagnosis of poisoning was made. Prognosis doubtful. Fluoroscopy was negative; no foreign bodies nor ulcerations were revealed. Mucous membranes of the mouth were cyanotic; even the animal's coat which had been a smooth, glossy tan had become drab. Breathing became faint and 1 cc. of metrazol was administered intravenously, after which respiration became stronger. Before further treatment could be instituted, the animal suffered a massive, fulminating oral hemorrhage and expired within a few minutes.

Case 5. Muggsy, mixed breed, seven years of age, weight 16 lb., male, who was also

exercised at the same place as the dog in case 1, became ill at noon on July 28. Bloody vomitus and tarry stools were reported. The patient did not appear acutely ill to the owner. However, on July 29, the dog was found dead in his bed. There had been a massive oral hemorrhage preceding death.

Case 17. Laddie, one-year-old Collie, male, weight 28 lb., became ill on August 20. The animal appeared to be in shock. Bloody stools and vomitus were noted. The mucous membranes were cyanosed, pulse was rapid, respiration shallow, temperature 100.2 F.

Two cc. of koagamin were injected upon admission, followed by 200 cc. glucose-saline into which 2 cc. koagamin had been incorporated. Two cc. vitamin K (2 mg.) were injected intramuscularly. The animal revived somewhat and an hour later 1 cc. koagamin was administered intramuscularly, followed by 150 cc. glucose-saline solution with 1 cc. koagamin. Four hours later the dog was pronounced out of danger. Sulfasuxadine, 1.5 gm., was administered orally and 150 cc. of milk were given with ten drops of brandy. The animal was discharged 36 hours later.

Case 38. Mac, two-year-old Scottish Terrier, male, weight 18 lb., became ill on August 22. The animal exhibited the same symptoms as all the other dogs in this series. Unfortunately, our supply of koagamin was exhausted, because of 16 poisonings treated between August 19 and 22. Saline-glucose solution 200 cc. and 2 mg. of vitamin K were administered. After one hour, the dog's respiration became labored and the heart action weak. Metrazol 1 cc. was injected intravenously and the animal's condition improved to some extent. However, 90 minutes after admission, the animal suffered a massive oral hemorrhage and expired. This was our first death since case 7 and the only one of these 30 cases to which koagamin had not been administered.

REFERENCES

1. Wisconsin Alumni Research Foundation Bulletin, 1951.

2. Klausman, B. S., The Use of a New Chemotherapeutic Agent to Arrest Bleeding in Animals. *Vet. Med.*, 46:282 (July), 1951.

3. Blamey, E. R., Coagulation in Seeping Hemorrhage—Evaluation of Koagamin, New to the Veterinary Field. *Jour. Am. Vet. Med. Assn.*, 119:291 (Oct.), 1951.

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The radial vein can be raised easily for intravenous injection by holding a rubber band in place with hemostats. Releasing the instrument immediately relieves pressure on the vein. —*R. E. Ruggles, D.V.M.*

Scrap Metals in Urgent Demand

The Defense Production Authority has repeatedly invited the attention of all businessmen and farmers to the urgency of getting scrap metals to mills. Shutdowns are anticipated because of critical shortages of scrap required for steel furnaces. Reduced output will immediately affect civilian production.

Many types of organizations have solicited the cooperation of members. These include those who may have accumulations of metal or are in position to influence others to check premises for scrap required. Tons of worn-out and discarded machinery are rusting on farms everywhere. This source of scrap must be salvaged, not added to junk yard inventory, and must be moved to points where it can be utilized. All have a vital interest in the campaign to "get in the scrap".

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Influence of Protein on the Milk Fever Complex

(*Continued from page 218*)

tion of Labeled Amino Acids into Tissue Proteins *In vivo* and *In vitro*. *Physiol. Rev.*, 30:206, 1950.

12. Geiger, E., The Role of the Time Factor in Protein Synthesis. *Science*, 3:594, 1950.

13. Cantarow, A., Diseases of Metabolism, edited by Children's Memorial Hospital, Chicago, 9:1697, Aug. 1950.

14. Best, C. H., and Taylor, N. B., The Physiological Basis of Medical Practice, ed. 4, Baltimore, The Williams & Wilkins Co., 1946.

15. Rawson, A. J., and Sunderman, F. W., Studies in Serum Electrolytes, XV: The Calcium-Binding Properties of Serum Proteins. *Jour. Clin. Inv.*, 27:82, 1948.

16. Albright, F., Note on the Management of Hypoparathyroidism with Dehydrotachsterol. *Jour. Am. Med. Assn.*, 112:2592, 1939.

17. Madsen, Louis L., Nutritional Diseases of Farm Animals, Yearbook of Agriculture, U. S. Printing Office, Washington, 1942, p. 327.

18. Hawk, P. B., Oser, B. L., and Summerson, W. H., Practical Physiological Chemistry, ed. 12, Philadelphia, The Blakiston Co., 1947, p. 546.

19. Phillips, R. A., Van Slyke, D. D., Dole, V. P., Emerson, K., Jr., Hamilton, P. B., and Archibald, R. M., Copper Sulfate Method for Measuring Specific Gravities of Whole Blood and Plasma, United States Navy Research Unit at the Hospital of the Rockefeller Institute for Medical Research, 1943-44, republished by Josiah Macy Foundation, New York, 1945.

20. Dukes, H. H., The Physiology of Domestic Animals, ed. 4, Ithaca, N. Y., Comstock Publishing Co., 1939, p. 49.

21. Moore, G. R., Bovine Acetonemia, Proceedings Book, Am. Vet. Med. Assn., Milwaukee, Wis., 1951, p. 43.

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See you at Atlantic City A.V.M.A. convention — June 23-26

Neomycin Sulfate In Veterinary Dermatology And Ophthalmology

J. LAVERE DAVIDSON,* D.V.M., Kalamazoo, Michigan

IN this age of wonder drugs, as they are now frequently designated, many new antibiotic substances have been made available to the practicing veterinarian. It is agreed that the treatment of certain animal diseases has been facilitated by these new drugs. Generally speaking, their use has been directed chiefly towards systemic effect. The use of antibiotic drugs topically, while employed widely, has not been acclaimed to the extent that the systemic application has been.

In 1949, Waksman and Lechevalier[1] reported a new antibiotic derived from the culture of a soil organism closely related to *Streptomyces fradiae*. Its clinical possibilities looked remarkably good because it exhibited activity against a wide variety of both Gram-positive and Gram-negative organisms. This new antibiotic was named neomycin.

While the chemistry of neomycin is still uncertain, it is postulated that it may have a cyclic acetal type structure. It is a polybasic compound, thermostable and soluble in water but insoluble in organic solvents. The sulfate salt is a white amorphous powder and in the dry form is stable for at least a year when stored at room temperature. Prepared solutions retain their potency for at least five months, although there is a progressive deepening of color in solutions stored at room temperature or 37 C. Refrigeration is, therefore, recommended for solutions of neomycin.

Unlike other antibacterial agents, it is extremely stable and very active in alkaline solution. Like streptomycin, it is assayed by the plate method against a standard. A Waksman unit of activity is defined as that amount in 1 ml. of broth or medium which will inhibit the growth of *Escherichia coli*. One milligram of the pure crystalline salt contains 200 units (±15%).

The following chart shows that *in vitro* studies demonstrate the effectiveness of neomycin in inhibiting representative specimens of both Gram-positive and Gram-negative organisms[2,3].

Antibacterial Spectrum of Neomycin

(Expressed in micrograms per ml. required to inhibit test organisms)

Str. agalactiae (15)	.17	*Ps. aeruginosa*	.33
Str. dysgalactiae (22)	.17	*Br. abortus*	.05
Str. uberis (29)	.17	*K. pneumoniae*	.10
Staph. albus	.01	*Proteus vulgaris*	.13
Staph. aureus	.01	*Sal. schottmuelleri*	.11
Str. fecalis	.11	*M. tuberculosis* (607)	.29
B. anthracis	.02	*D. pneumoniae*	10.00
A. aerogenes	.14	*Str. viridans*	33.00
E. typhosa	.03	*Str. hemolyticus*	10.00
E. coli	.11		

It is easily understood that any drug such as neomycin, which has a broad antibacterial spectrum and is suitable for external use, would be a welcome therapeutic addition to veterinary dermatology and ophthalmology.

During the past two years, the writer has used this agent in veterinary practice in the treatment of many skin, ear, and eye infections with remarkable success. The preparations used are an ointment, myciguent®, containing 5 mg. neomycin sulfate per gm., both as a general surgical ointment and an ophthalmic ointment, and an aqueous solution.

These preparations have been given to over 300 animal patients suffering a variety of clinical abnormalities. Many of these had been treated with other medicaments and were refractory but did respond to this therapy. In some cases, positive laboratory diagnoses as to the bacterial organism involved were accomplished, but the majority were treated without benefit of laboratory aid.

Myciguent was employed initially for the treatment of otitis externa which had not responded to other forms of therapy, particularly cases that were caused or complicated by Pseudomonas organisms. These Gram-negative bacteria have proved more or less refractory to other antibiotic agents. The results with myciguent have been unusually good.

In several types of dermatitis in both small and large animals, topical application of this

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Representative Cases Treated with Neomycin

Species	Diagnosis	Laboratory Findings	Previous Therapy and State of Illness	Results with Neomycin
Dog	Otitis externa (chronic)	Staphylococcus	Recurrent infections for over 1 yr. Treated with sulfas, penicillin and bacitracin.	Improved in 4 da.; recovered 2 wk.
Dog	Otitis externa (chronic)	Pseudomonas	Condition present for 3 mo.	Ears were cleared in 5 da.
Dog	Pustular eczema	None accomplished	Acute, had been treated at home for about 1 wk.	Improved rapidly
Cat	Otitis externa and media	None accomplished	Chronic for 4 to 5 mo. Had been treated with numerous other preparations.	Ears improved in 3 da. and were dry and healed in 3 wk.
Dog	Otitis externa (acute)	Pseudomonas	Condition present only a few days. No previous treatment.	Exudate checked in 2 da. Complete relief 1 wk.
Horse	Dermatitis	None accomplished	Condition present for about 2 wk. Had been treated locally with bacitracin, aureomycin and Burrow's solution.	Cleared in 10 da.
Cows (8)	Pink-eye	Not done	Acute	Recovery 48 hr.
Dog	Conjunctivitis	Not done	Chronic; condition present for about 2 wk. Had numerous other treatments.	Improved in 2 da.
Dog	Keratitis	Not done	Condition present for about 1 wk. Had been treated with penicillin ointment.	Recovery 1 wk.
Cat	Severe keratoconjunctivitis	Not done	Acute; no accurate history.	Improved in 1 da., cured in 5 da.
Cat	Opacity of cornea	Not done	No previous treatment.	Cornea clear in 4 da.
Dog	Pustular dermatitis	Staphylococcus and Pseudomonas	Condition present for 1 mo. Numerous topical preparations had been used.	Improved in 1 wk. Cured in 2½ wk.
Dog	Interdigital eczema	Staphylococcus	Chronic, many other medicaments had been used.	After drainage was established surgically, the lesions healed in 10 da. using neomycin wet packs.
Puppy	Impetigo	Staphylococcus	Acute; Burrow's solution had been used for about 3 da.	Cleared in 6 da.

antibiotic has proved effective, especially whenever pyogenic organisms were involved.

Ophthalmic ointment is recommended for the treatment of keratoconjunctivitis, ulcers of the cornea, corneal opacities, and in acute pink eye of cattle.

Application

Myciguent was used in most instances by applying directly to the affected areas, either with or without bandages or dressings, one to three times a day. If the lesions were moist or exudative, treatment was started by using wet dressings of aqueous neomycin solutions.

Ophthalmic ointment containing the new antibiotic was applied three times a day by expressing a small amount of ointment under the eyelid and massaging gently. This unguent melts and spreads rapidly over the entire ocular area and can be applied with ease.

No evidence of irritation was noted following application to any of the animal patients and no instances of sensitivity were recorded.

Conclusions

Neomycin sulfate has proved to be a superior antibiotic for topical use in treating a wide variety of skin, ear and eye infections in both large and small animals. During the past two years, over 300 animals have been treated with either ointment or an aqueous solution of the agent. A few representative cases have been charted.

Neomycin sulfate fills all of the requirements for a safe, effective means of treating many dermatological and ophthalmological disease conditions common in veterinary practice. It is especially effective in infections associated with Pseudomonas infection. Anomalies which had stubbornly resisted other means of therapy were treated successfully.

REFERENCES

1. Waksman, S. A., and Lechevalier, H. A., *Science*, 109:305, 1949.
2. Johnson, L. E., and Colingsworth, D. R., Upjohn Research Laboratories, unpublished data.
3. Drury, A. R., Michigan State College, East Lansing, personal communication.

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Canine Coccidiosis

(*Continued from page 228*)

13. Morgan, Banner Bill, and Hawkins, P. A., Veterinary Protozoology, Minneapolis, Minn., Burgess Publishing Co., 1948, p. 81.
14. Question and Reply Column. *N. Am. Vet.*, 29:310, 1948.
15. Craige, A. H., Jr., and Kleckner, A. L., Teniacidal Action of Di-Phenthane 70. *N. Am. Vet.*, 27:26, 1946.
16. Blair, H. E., Vermiplex, a New Anthelmintic for Dogs. *N. Am. Vet.*, 30:306, 1949.
17. Burch, G. R., and Blair, H. E., Vermiplex, an Anthelmintic for Cats. *N. Am. Vet.*, 31:329, 1950.
18. Ryff, J. F., *et al.*, Removal of the Fringed Tapeworm from Sheep. *Jour. Am. Vet. Med. Assn.*, 115:179, 1949.
19. Ryff, J. F., *et al.*, Removal of the Fringed Tapeworm from Sheep. *Jour. Am. Vet. Med. Assn.*, 117:471, 1950.

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Streptococcic Infections in Pigs

(*Continued from page 220*)

Streptococcic Mastitis

Mastitis in sows causes considerable loss of pigs, and of an occasional sow. Streptococci are associated with the udder lesion in many herds, and pigs often die with a generalized streptococcic infection when nursing such sows. Finding the infection in the dead animals has brought the udder condition to the attention of the veterinarian in many cases. Usually, the sow is lethargic, with a hot, sensitive, swollen udder. She will refuse to nurse pigs, or milk secretion is markedly reduced. Treatment depends on the cause. Care of the pigs is paramount.

Streptococcic Metritis

Metritis is one of the causes of infertility in sows and streptococci play an important part in this problem. If some of the affected animals breed, they would be possible sources for navel infection of pigs farrowed.

Tonsillitis

Tonsil infection is common in swine and streptococci play an important role in this process. A diagnosis usually is made at autopsy.

Wound Infection

Lesions about the head of small pigs, the result of fighting, may become infected and suppuration and necrosis may result with considerable distortion. Streptococci assume an important secondary role in many of these cases. The same holds true with wounds in general, as they provide a portal of entry for various infections.

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Offerings of larger numbers of slaughter hogs and lambs have been a major factor in lower prices.

Canine Pathology: An Historical Essay On Some Pioneer Contributions Of D. P. Blaine (1770-1845)

J.R.M. INNES,† Sc.D. (Cambridge), D.Sc. (Edin.), Ph.D., M.R.C.V.S., F.R.S.E.,
San Antonio, Tex.

"Some dispositions evince an unbounded admiration for antiquity, others eagerly embrace novelty; only a few can preserve the just medium, and neither tear up what the ancients have correctly established, nor despise the just innovations of the moderns."—Francis Bacon.

MANY years ago, because of an interest in the history of medicine, the writer was accused by an eminent medical worker of evincing the inevitable index of early senility. No scientific worker can ignore history, for after all, what is news one day is history very soon after. In scientific articles of today it may no longer be necessary to review steps in progress of even 50 years ago; yet we should avoid giving students the impression that, for example, ideas on infection arose only by the epoch-making discoveries of Pasteur and Koch. It might also be a revelation to young veterinarians to be aware of what was known about canine diseases to their forebears of 150 years ago.

Veterinary medicine has still to produce a Garrison, Allbutt, or Singer, among many others who have written in English on aspects of the history of human medicine. Veterinary science is young, and there may not be so much to write about, for it took the veterinary art a long time to arise from the stench of quackery and ignorant empiricism to the level of a science. Major General Sir Frederick Smith,* K.C.M.G., C.B. (1857-1929), one of the most distinguished British veterinarians, during the latter part of his life compiled a classical work dealing with, The Early History of Veterinary Literature and its British Development. This is the only work of its kind in English, and might be full of interest for those who have leanings in the historical direction. It is written around personalities and not, however, around chronological developments on the history of progress in the understanding of animal diseases. From his volume III, most of the notes on Blaine's life have been paraphrased, although Blaine, in the introductions to some of his books, recounts some of his own life in a quite unblushing manner. In truth, the name of William Youatt (1776-1847), who was associated as a partner in practice with Blaine for a few years, and his works should not be disassociated, but to consider both in the one article would make for complexity. Smith referred to Youatt as a gentle and amiable man of great benevolence and humanity, a prodigious worker, a pioneer in veterinary science and devoted to its progress. In many ways he was a greater man than Blaine, and certainly was never a braggard.

Apprentice Surgeon—Anatomist

Delabere Pritchett Blaine, at 14 years, was apprenticed to a surgeon, with whom he served seven years before proceeding to the Borough Hospital in London (then united as Guys and St. Thomas'). During this period, he apparently did some work on regeneration of nerves, and on finishing served as teacher of anatomy, with no knowledge of the anatomy of the horse, at the newly established Veterinary College in London. He left because of personal quarrels with Sainbel (the head of the College) and started in veterinary practice. Smith says he must have been clearly unfit for practice, far less qualified than the cow-leech or farrier of that time. Like many, however, he believed that "a training in human medicine would see him through, but Blaine did not realize then that there was something which a study of human medicine could not teach regarding diagnosis of disease in dumb brutes."

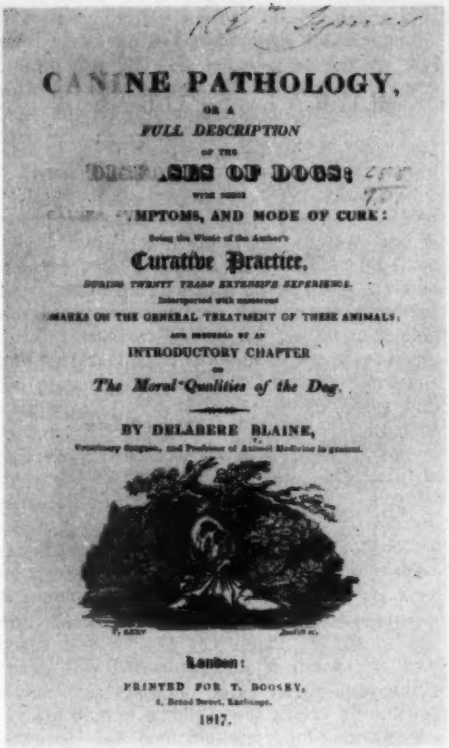
He joined the Army in 1793 as a surgeon, resigned in 1795 to begin a medical (surgical) practice in London, but says he was much occupied in making drawings for a book on the

†Foundation of Applied Research, San Antonio, Tex.

*Smith, between 1876-1929 wrote a manual on Veterinary Hygiene (1887); a manual on Saddles and Sore Backs (1891); a Manual on Veterinary Physiology (1892), which ran to the 5th edition in 1921; and over 100 articles on a fantastic variety of subjects covering anatomy, histology, physiology, pharmacology, pathology and clinical subjects, apart from his notable efforts on the history of veterinary anatomy, and of the role of the Army Veterinary Corps in the Boer War and World War I. A biography of Smith was written by Fred Bullock—vol. IV of Smith's work.

anatomy of the horse, and seemingly he was no mean draftsman. He then tells that he came into a considerable fortune which induced him to retire into the country, but candidly admits he had not gained a prudential mode of managing money, and after living expensively as a country gentleman for a few years found it necessary to enter active life. Perhaps it was this period which gave him opportunity to gain an invaluable intimate experience of the habits of domestic animals, and everything connected with their use in sports. In an effort to recoup his fortune he compiled, according to Smith, a disgraceful work of self-advertisement, dealing with a so-called efficacious remedy for distemper. The less said about this production the better; doubtless he was later ashamed, for in 1841 he wrote that "There is not, and cannot be, a specific for the *protean* disorder" (distemper), words which are still true in 1952. He returned to veterinary practice around 1801 in London, and must have found it lucrative. He wrote An Anatomy of the Horse, A Treatise of the Diseases of the Horse and Dog, and a second edition of his shameful quack work—which Smith says robbed him of the dignity with which his name should be wholly clothed. As a result he was rightly ostracized by the veterinary profession; a blow which he bitterly felt all his life.

He took Youatt into partnership, and apparently this lasted until about 1817. In that year, he published his first edition of Canine Pathology (the frontispiece illustrated), and had he compiled no other work it would have placed him in the company of the pioneer select. He had been trained in the experimental method of medical enquiry, which enabled him to make many shrewd observations and descriptions of disease in animals, some of which indeed have hardly been improved upon in the 134 years which have elapsed since. This will be apparent later from quoted excerpts of his writings. Although in his book he mentions few eminent medical men of that period, in virtue of his training and wide reading he must have been greatly influenced by the blooming of knowledge on human morbid anatomy and clinical medicine so apparent about 1800.


CANINE PATHOLOGY,

OR A

FULL DESCRIPTION

OF THE

DISEASES OF DOGS;

WITH THEIR

SYMPTOMS, AND MODE OF CURE:

Being the Whole of the Author's

Curative Practice,

DURING TWENTY YEARS EXTENSIVE EXPERIENCE.

Interspersed with numerous

REMARKS ON THE GENERAL TREATMENT OF THESE ANIMALS;

AND PREFACED BY AN

INTRODUCTORY CHAPTER

ON

The Moral Qualities of the Dog.

BY DELABERE BLAINE,

Veterinary Surgeon, and Professor of Animal Medicine to ground.

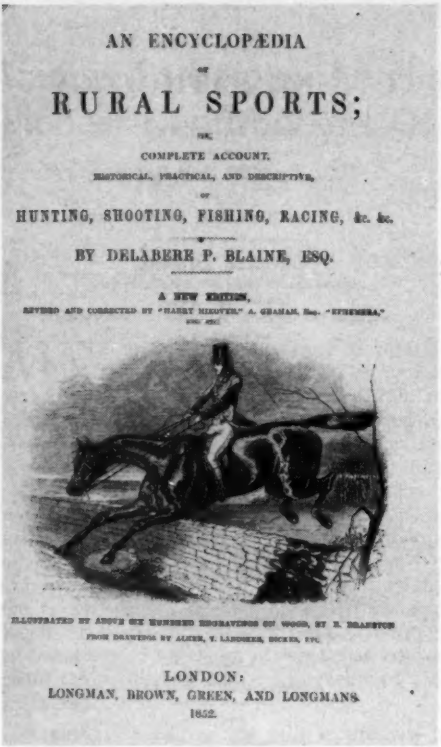
London:

PRINTED FOR T. BOOSEY,

1817.

Contemporaries of Blaine

The early part of the 19th century was the eve of great medical discoveries. Much was known by then about the structure of the human body; Morgagni had paved the way for future pathology and a new spirit was being adopted towards disease investigations, which was leading at least to a better understanding of the relationship between symptoms and structural changes. Bichat (1772-1802) and Laennec (1781-1826) had already made their memorable contributions to pathology; the latter having left masterful descriptions of emphysema, lobar and lobular pneumonia, and bronchiectasis correlated with the clinical signs elicited by percussion and auscultation (for he had discovered the stethoscope); Laennec also realized that all the various manifestations of tuberculosis (tubercle to cavity) were parts of a single process. Blaine also lived in London, where the famous William and John Hunter had achieved immortal fame, and where Matthew Baillie (1761-1823) had already published the first pathological atlas in English (1792) which demonstrated morbid anatomy to the medical student. Almost contemporary with Blaine in London there were Bright (1785-1855), kidney disease; Addison (1793-1860), pernicious anemia; and Hodgkin (1798-1866), lymphadenoma. He must have been aware of all the rapidly rising trends, and the new enthusiasm for postmortem work.


AN ENCYCLOPÆDIA

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OR,

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HISTORICAL, PRACTICAL, AND DESCRIPTIVE,

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LONGMAN, BROWN, GREEN, AND LONGMANS.

1852.

In the case of the dog, Blaine was not faced with the medical problem of public antipathy to autopsies. Thus, in his book on canine pathology he emphasized the great necessity for postmortem examinations of animals, of which he made many hundreds on cases of rabies alone. He claimed to have seen more cases of rabies than any man then alive. His comments on drugs were very precise. Smith thus says, "Can it be doubted if our veterinary school (London) had carried out these lessons to be learnt from experimentation and post-mortem exactitude, we should have been spared the long years of guessing and worthless conjecture which did so much to retard the growth of our profession." As it was, we had to wait more than 80 years, and the slough of despond of veterinary pathology in English speaking countries has only been left behind within living memory.

Blaine seems to have had a remarkable memory, to have read widely with pertinacious criticism, and to have had a special aptitude for history. With this, one would imagine, apart from his knowing about the floodtide of new medical knowledge coming from the London, Edinburgh and Dublin medical schools, he must also have been fully aware of the tremendous events taking place in Germany and France which were to lead to Virchow. Apart from his book on Canine Pathology, he published an original, inimitable volume called An Encyclopaedia of Rural Sports (1840) (frontispiece illustrated) which he says occupied him *"seven years indefatigable labour."* As a reader, one could never doubt this, for it is reputed to contain some one and a half million words, contained in 1,247 pages (1851 edition in possession of the writer), and in the most minute, but readable, print, with hundreds of little engravings. This deals with every conceivable aspect of sport in England of the early 19th century, all animals (their history, origin, anatomy, physiology, diseases) and the sport itself; horses are dealt with at length, including racing, training, jockeyship and book-making; information concerning all angles of human activities in the hunt, field and stream can be found. This work alone stamps him as a man of magnificent talent as a scientist, scholar and sportsman.

Canine Pathology—a Remarkable Work

We are not concerned here with his other books, but only with the pioneer work on Canine Pathology. It was first published in 1817, and other editions appeared in 1821, 1824, 1832, 1841 and one (1851) after his death. It was published by Mayer, London, and was translated into French, German and Italian. He claims (rightly) in the first edition, that he was the first person in England "who has systematized and brought forward a regular medical treatment of the diseases of these animals founded on a knowledge of their anatomy and economy with a long attentive observation of their morbid appearances." He claims that the whole work was his own, "not a line having been previously written by any other hand." It is amusing to note also he says, "Having been educated as a human surgeon . . . I greatly offended my relations and surprised my friends and acquaintances, that I should degrade myself by the study of the disease of animals." Here we see the result of an attitude which has taken a long time to dissipate, and even yet rears its head. Starting with the Greeks, veterinary activities were regarded as a contemptible occupation and beneath the dignity of educated man. Man's concepts of his own scientific obligations were largely anthropocentric; the "horse doctor," or "vet" in the minds of many had always occupied a lowly place, and medicine was not

entirely without blame in this direction. It was considered highly intellectual to study diseases of the prostate of man, but not of the brain of the sheep or pig. Yet plant pathology has always been scientific.

Some of Blaine's most notable observations might be dealt with at length. He, and Youatt, were champions in the belief of the specific nature of rabies, one far removed from a general view held both by medical and veterinary men of that period, who held Blaine and Youatt up to ridicule. Smith mentions that nevertheless they were not the first, for Doctor Bardsley (England) in 1793 had enunciated in print this same theme. Smith quotes a view of Bardsley, which is startling in that he advocated that rabies could be controlled by a total quarantine of all dogs in England for a period of two to three months; a view that took nearly another century to establish.

There are 36 pages of splendid clinical description, although remarks on morbid anatomy are still in a relatively primitive stage. He insists that rabid dogs instead of showing a dread of water (hydrophobia), in most instances seek it with avidity, and may be found "still anxiously hanging over the liquid even if they cannot swallow." He alludes to the very old, popular fallacy of its being due to "worm under the tongue." He says, "No dog breeds madness . . . no dog becomes mad from any other cause whatever but his being bitten or being inoculated by another." He asks, "How came the disease in the first place? In answer, how came human smallpox, measles or syphilis?—all at first generated by combinations unknown to us, but they are never now produced but by infection . . . nothing short of inoculation by the bite of a rabid animal (probably of dog and cat only or their kinds), or any insertion of the rabies virus into a wound or abraded surface." (It was not until 1903 that Remlinger identified a filtrable virus.) His remarks on clinical signs are of renown, for he mentions the peculiarity of the dog's manner . . . appetite is not always lost . . . violent scratching, licking or even biting of some part of the body . . . the great irritability yet "meekness may continue to the last." He observed, "When the throat is much affected, the jaw paralytic and the tongue pendant, there is a choaking noise." He noted the difference between the raging and dumb types, and he elaborated on the propensity of the affected animal to rove. Then he states that few die sooner than the third day, very few survive the seventh, while his estimate on incubation period was that the majority showed symptoms between the third and seventh week, but in many cases three or four months elapsed. He believed no remedy was known, and that the disease was universally fatal, although treatment of the affected bitten part was advocated by him.

There are many outstanding observations dealing with a variety of other visceral diseases. On asthma he noted (rightly so) that it is different from the human disease, and in the dog "was dependent on morbid and fixed alteration in the structure of the lungs" (which was not, of course, true asthma). In two or three instances he found "a considerable tumour attached to the root of the diaphragm, the pressures of which caused the asthmatic symptoms" (no doubt tuberculosis of the pulmonary nodes, or true tumour). He mentions that schirrhous tumors were very common in the mammae and teats of bitches . . . and in testes of male dogs, and he recommends in the latter type "to proceed without delay to castrate to prevent the disease from extending up the spermatic cord." On every page there is something which brings to mind his keen eye, if one can visualize the general state of civilization of 150 years ago. When he discusses the disease distemper, the writer concludes that there is hardly any phase of the clinical picture which is not almost completely covered. His contribution† could replace descriptions in many modern veterinary textbooks, and there would be little lost that is of value. Whatever else this does, its reading should teach us some humility, for in the past few years there has been a tremendous recrudescence in interest in distemper-like diseases of the dog. It is clear that in the veterinary textbook literature subsequent to Blaine, like many medical sciences, there has been a tendency to transfer quite uncritically data from textbook to textbook and as far as distemper is concerned this must be notoriously apparent.

The foregoing essay might serve as a number of lessons for students and others. First, history by itself, no matter on what subject, has an intellectual value, and one wonders whether it is opportune for veterinary schools to follow the example of many medical ones and introduce a short course into the veterinary education curriculum, *not for examination purposes.* In the realm of animal medicine, it serves to demonstrate what was achieved a long time

(*Continued on page 258*)

†A full transcript of Blaine's description of distemper will appear in a later issue.

EDITORIAL COMMENTS

Research and Disease Control Programs Intensified

More defense jobs and a steady growth in population are increasing the demand for meat and other animal products. Because of these greater needs, the research and disease control programs of the BAI are being intensified to increase the efficiency of livestock production and safeguard animal products so vital to the country's defense plans.

In 1951, reports Dr. B. T. Simms, Bureau chief, special measures were taken to protect farm animals from diseases which might be introduced from abroad. Added precautions have been taken at the borders and ports of entry in the inspection and quarantine of animal imports.

Provisions of the Tariff Act of 1930, prohibit importations of domestic ruminants and swine from countries in which foot-and-mouth disease and rinderpest exist. The same prohibition holds for the importation of fresh, chilled, or frozen beef, veal, mutton, or pork from those countries.

As a further safeguard, the second in a series of training schools was held to familiarize additional Bureau veterinarians with the symptoms of serious foreign animal plagues. Thus, with the help of well-trained veterinarians located in each geographical area, it would be possible to detect and eradicate disease before it can spread to other areas. Also, ten regional conferences with state and Bureau veterinarians resulted in plans for organizing all segments of the veterinary profession and livestock industry as a means of preparing for early detection and proper reporting of any suspected animal disease outbreak.

Concerning the control of diseases already prevalent, the report shows a further reduction in the incidence of brucellosis to 3% of all animals tested; approaching the ultimate goal of complete eradication. Also, there was a reduction in the incidence of bovine tuberculosis. Constant alertness on the part of Bureau and state veterinarians thwarted a widespread invasion of the range states by sheep scabies.

Poultry nutrition studies revealed that when small amounts of antibiotics were included in the ration of growing chickens, 19% protein was as effective as a higher percentage. Without the antibiotic, 21% protein was needed. Although the difference between these two percentages is small, a similar reduction in protein content in all the starter and broiler mashes used in the United States would result in an annual saving of 125,000 tons of protein.

During the period covered by the annual report of the Bureau, this agency certified more than 27,000 breeding animals for importation, a slight increase over the number recorded last year. The figure includes 21,940 cattle of various breeds, 3,341 sheep, and 917 hogs. Under the provisions of the Tariff Act of 1930, breeding animals may be imported duty-free when they can be certified as purebred.

Details of its various regulatory and research activities are included in the 91-page report which may be obtained by writing to the Bureau of Animal Industry, U. S. Department of Agriculture, Washington 25, D. C.

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New York Legislature Passes Bill to Provide Pound Dogs for Research

The vigorously opposed Hatch-Metcalf animal research bill was signed by Governor Dewey of New York and became state law on March 10, 1952. The bill makes possible the requisition of doomed, unwanted, and homeless dogs and cats for use in medical research and teaching.

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DDT-Resistant Insects, a Warning Against Dependence on Insecticides Alone for Control

Tests conducted during the past year in heavy mosquito-breeding areas of Florida and California have indicated that mosquitoes are more resistant to DDT than formerly. They were not noticeably resistant to newer insecticides, including BHC, lindane and chlordane, but are likely to become so as these come into more general and extensive use.

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The population of the world has increased by 1.2 millions in one century, passing from 1,171,000,000 in 1850 to 2,378,000,000 in 1949. Since 1650, the number of world inhabitants has been nearly quadrupled.—*Chron. WHO.*

Insect Pest Survey and Reporting Service

A cooperative insect pest survey and reporting service, in which entomologists associated with state agencies, industry, and the federal government will participate, is announced by the USDA. The purpose is to keep the nation's farmers informed of impending insect conditions. The service will attempt to forewarn farmers, no matter what crops or livestock they produce, of current insect pest conditions, and will allow them to meet these threats better.

Defense-wise, the survey organization will provide the skeleton organization needed for protection against biological warfare if the occasion arises.

Entomologists and other workers engaged in agricultural pursuits will cooperate to make this new plan work. It is planned that any insect information within a state, whether detected by federal, state, local or commercially engaged entomologists, will be channeled through state offices so that the information can be released quickly to meet local requirements. The report will then be forwarded to Washington, D. C., where a Section of Economic Insect Detection and Reporting in the Bureau of Entomology and Plant Quarantine will combine the information and distribute it nationally.

This service gives promise of great value, not only to individual growers but to control agencies concerned everywhere. If only a small portion of savings is realized, the venture will be economical. Hopefully, this may prove to be forerunner to an organization for collection of vital statistics of animal diseases which are needed for accurate estimates and forecasting threats to livestock health. Much such information will be valuable to veterinary practitioners.

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One of the most useful laboratory tests in our practice is the blood urea determination (La Motte method). It is most useful in the differential diagnosis of leptospirosis in dogs. Increase in blood urea is observed concurrently with the first clinical signs of the disease. In mild cases there is often a rise of 60 to 80 mg. Such patient exhibits only slight symptoms of illness and in two or three days, blood urea will return to normal and the condition of the dog will parallel test findings.—*Charles E. Bild, D.V.M.*

Edema Disease of Swine

Three factors are common to every case of edema disease in pigs with which the writer has dealt: (1) All have been fed a wet ration; swill prepared with little meal and a large quantity of water; (2) the anomaly occurs most frequently in "backyard" pigs; (3) it is uncommon to find a single animal in a drove succumbing to this condition.

Treatment has not been encouraging. The disease, resembling essentially an allergy, does not respond to antihistamine therapy. Recoveries have been effected by starvation for 24 to 48 hours, during which time heat and dry quarters are provided and stimulants given. Strong solutions of magnesium sulfate are prescribed, after which dry bran is offered. Water is withheld until the fifth or sixth day of treatment. Parenteral vitamin B appears to be effective in mild cases. No treatment is of any avail during advanced stages of the disease.—*William A. B. Barr, Vet. Rec.*, 64:168 (Mar. 15), 1952.

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Fireweed Poisoning

The California State Department of Agriculture believes that recent heavy losses of cattle in the counties of San Luis Obispo, Monterey, San Benito and Kern were due to the animals' eating damaged fireweed in which potassium nitrate had been built up well in excess of the toxic limit.

Field surveys in the areas from which the deaths were reported indicated that this year the animals had eaten the weed extensively. When other animals in the same herds were taken off the pasture, put in corrals and fed hay, deaths ended.

Cattlemen reported that fireweed has been common for many years in the area; usually cattle do not eat it and therefore it has not caused trouble. This year, however, some chemical change apparently occurred in the plants, perhaps due to climatic shock, which caused them to build up nitrate sufficient to cause symptoms of poisoning and deaths.

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Trichomonad-like organisms were reported in smears made from scanty mucopurulent nasal exudates of two pigs. Similar protozoan organisms were isolated from infected animals in a recent outbreak in Michigan involving three herds which, on autopsy, revealed advanced symptoms of atrophic rhinitis.—*M.S.C. Vet.*, Winter, 1952.

Dr. I. A. Merchant Named Dean of Veterinary Medicine at Iowa State College

Dr. I. A. Merchant, head of the Department of Veterinary Hygiene at Iowa State College, will become dean of the Division of Veterinary Medicine and director of the Veterinary Research Institute, July 1, 1952.

Dr. Charles E. Friley, president of the college, said that Merchant's appointment had

Dr. I. A. Merchant

been approved recently at a meeting of the State Board of Education at Ames. On July 1, Dr. H. D. Bergman, who has served as dean since 1943, will relinquish his administrative duties, and devote his time to teaching and research.

Doctor Merchant was born in Colorado, and received the degree of Doctor of Veterinary Medicine from Colorado State College in 1924. After spending 1½ years with the U. S. Bureau of Animal Industry in Nebraska he came to Iowa State College as instructor in veterinary pathology, and became assistant professor in that department in 1928.

Upon the formation of the Department of Veterinary Hygiene in 1930 he was transferred to that Department, teaching bacteriology and food hygiene. Doctor Merchant was made associate professor in 1934 and became professor and head of the Department in 1943.

He received the Master of Science degree from Iowa State College in 1928 and the Doctor of Philosophy degree in 1933. He spent the year 1933-1934 in postdoctoral study in the Department of Public Health of Yale University College of Medicine.

He is author of the textbook, Veterinary Bacteriology and Virology, which is used in many of the veterinary colleges in the country, and is now in its fourth edition. A laboratory manual, which he wrote with R. A. Packer, also is used widely. In 1951, he wrote An Outline of the Infectious Diseases of Animals which has been adopted also by many veterinary colleges.

He is a member of the Iowa Veterinary Medical Association, and the American Veterinary Medical Association, as well as one of the founders of the American Board of Veterinary Public Health. He is a member of Phi Kappa Phi, Phi Zeta, Alpha Psi, Sigma Xi, and Delta Omega honor societies; of the Osborn Club; of Sigma Nu social fraternity, and past president of the Ames Rotary Club. He is a member of the Protestant Episcopal Church.

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"Meat on the Hoof"

America's "Meat on the Hoof" is the title of a 38-page article, illustrated with colored reproductions, in the January 1952 issue of *The National Geographic*. This survey features the fact that the livestock raisers are trying to please the customer, the American housewife, with small roasts, leaner pork chops, and bigger-breasted fowls. The breeding programs are patterned to the more merchandisable carcass and the stock show judges are selecting as winners those that conform to current market trends. The survey indicates that about 6% of the take-home pay in America is budgeted for meat food products and that about 30% of the total farm income is from meat animals.

St. Paul, Minn., is rated America's largest calf market. Iowa is rated the largest feeder cattle state. In many localities, 20% of the income of the dairy farmer is from the sale of his animals for meat. It is stated that one-third of all the pigs born in the United States die before they are weaned.

Many colored pictures of the King Ranch operation in Kingsville, Tex., are reproduced. The Santa Gertrudis cattle at this location are recognized as the only breed developed in America, a cross about three-eighths Brahman and five-eighths red Shorthorn. The cattle are deep red, droopy-eared, horned cattle, with desirable beef conformation. They dress out 2 to 3% higher than other beef brands, withstand the hot sun of southern Texas, are more resistant to insects, and are practically free from cancerous eye. Incidentally, other states and central countries (Cuba, Australia, etc.) are buying Santa Gertrudis bulls at fancy prices; one sale of 29 averaged about $3,400.

The Minnesota State Experiment Station is credited with development of two distinct breeds of hogs; Minnesota no. 1, crossing Danish Landrace with British Tamworth, and Minnesota no. 2 by crossing Poland China with Yorkshire. The Idaho Experiment Station is credited with development of an American sheep breed, the Columbia, crossing the Lincoln and Rambouillet.

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New Foot-and-Mouth Disease Outbreak in Canada

Alert Canadian inspectors found fresh lesions of foot-and-mouth disease in a herd of 41 cattle on April 28, 1952. This outbreak was 28 miles outside the original quarantine zone, approximately 75 miles southeast of Regina, and 50 miles from the United States border. Preparations were made immediately to slaughter and bury infected and contact animals. Quarantine area was extended to include the new area of infection. To May 5, three Weyburn district herds have been condemned. Slaughter of infected animals brought the Weyburn district aggregate to 248 cattle and 162 hogs and southern Saskatchewan's total to 1,742 since the first infection was reported February 25.

Sporadic outbreaks of this nature are to be expected for some months, according to Bureau officials. The importance of prompt identification and restriction of livestock movements in infected areas is well recognized by Canadian veterinarians. Activities including herd inspections and strengthening quarantine measures are being intensified.

The BAI took further steps during the past month to safeguard against introduction of the disease in this country by tripling the number of inspectors along the northern boundary of Montana and North Dakota. These men, 33 in number, are assigned to a mobile border guard.

Recent news releases advise that BAI inspectors working in cooperation with various state livestock sanitary officials, have completed observations of animals brought into this country from Canada during the period just prior to closing the border to importations. These fieldmen report that over 5,500 cattle and approximately 250 hogs and sheep have been located at 138 points in the United States. Animals were traced and observed. No evidence of infection has been revealed.

American Animal Hospital Association, Pasadena Meeting

A total registration of 571, including members and guests, expressed unanimous enthusiasm for the expertly presented program of the 19th annual meeting of this group, held April 30-May 2, 1952. Excellent facilities of

Dr. Myron A. Thom

the beautiful and spacious Huntington Hotel in Pasadena added to the enjoyment of all those in attendance.

Special features were three television sessions. Bright 19 inch screens were placed on the rostrum and spaced at intervals of approximately ten feet along both sides of the hall. One had a "front row" seat at any location in the large meeting room. Surgical technics and other demonstrations, including laboratory and *x*-ray procedures, were televised from the Thom Veterinary Hospital in downtown Pasadena. Every detail was clearly shown on the screen. Explanatory remarks by the surgeon and moderator set a new high for clarity of demonstrations. Dr. Wade O. Brinker, Michigan State College, East Lansing, acted as studio moderator for two sessions and Dr. W. W. Armistead, Texas A. & M. College, College Station, for the third. Demonstrating surgeons included Gerry B. Schnelle, Boston, Mass.; Reginald A. Stocking, Los Angeles, Calif.; Harlan E. Jensen, Cleveland, Ohio; Ivan C. Frederickson, Hollywood, Fla.; James H. Yarborough, Miami, Fla., and William Magrane, Mishawaka, Ind. Demonstrators were Robert P. Knowles and Charles E. Bild, Miami, Fla., and Myron A. Thom, Pasadena, Calif.

Not all the time was devoted to television sessions. A wide range of papers was presented by national authorities.

Dr. Myron A. Thom, Pasadena, new president, succeeded Dr. Ralph E. Ruggles, Moline, Ill. Dr. Louis A. Corwin was advanced to president-elect and Dr. Charles W. Bower, Topeka, Kan., formerly president of the A.V.M.A. was elected vice-president. Dr. Arthur R. Theobald, Cincinnati, Ohio, was re-elected treasurer and Dr. Wayne H. Riser, Skokie, Ill., retained as executive secretary.

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Raymond A. Kelser Succumbs to Heart Attack

Raymond Alexander Kelser, D.V.M., M.A., Ph.D., Brigadier General, U. S. A. retired, died suddenly on April 16, 1952, following a heart attack with which he was overcome while at work. He apparently had been in good health up to the time of the seizure. His age was 59.

At the time of his death, Doctor Kelser was dean of the School of Veterinary Medicine and Professor of Bacteriology at the University of Pennsylvania. He was formerly chief of the Army Veterinary Corps and one of the country's leading bacteriologists and immunologists. He was widely known for his work in developing a greatly improved vaccine for rinderpest, while serving on the Medical Department Research Board in the Philippines 1927-1928. The large scale use of this vaccine enabled the Philippine government to eradicate the dread "cattle plague" from the Islands. Doctor Kelser was also renowned for his discovery of the mode of transmission of equine encephalomyelitis. He had been an active investigator of problems of animal disease, especially concerning himself with the filtrable viruses, rabies, encephalomyelitis, and Chaga's disease.

Doctor Kelser received his D.V.M. from George Washington University in 1914. He was awarded an A.M. (1922) and a Ph.D. (1923) from American University in Washington. He was commissioned first lieutenant in the Army Veterinary Corps in 1918. He helped organize and later commanded the first army veterinary laboratory at Letterman Army Hospital in San Francisco. In 1938, the then Colonel Kelser was appointed chief of the Veterinary Corps and served in the Office of the Surgeon General in Washington in that capacity throughout World War II. During his tour of duty as chief he was promoted to the rank of brigadier general, to be the first general officer of the Veterinary Corps.

Selection of Doctor Kelser as dean of the Veterinary School at the University of Penn-

Brigadier General R. A. Kelser

sylvania was made in 1946. He succeeded Dr. George A. Dick, who had asked to be re'ieved of the executive post.

Doctor Kelser was the author of numerous scientific papers on veterinary and bacteriological subjects. In collaboration with Dr. Harry W. Schoening of the BAI, he published the standard reference work, Manual of Veterinary Bacteriology, now in its fifth edition. He was the holder of the Distinguished Service Medal and the International Veterinary Congress prize; the latter awarded him in 1942 by the A.V.M.A. In 1949, he received the diploma of Honorary Associate of the Royal College of Veterinary Surgeons in London, together with two other American veterinarians.

He is survived by his widow, *nee* Eveline Davison, and one daughter, Mrs. John A. Allgair.

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Rumors of new outbreaks of foot-and-mouth disease in Mexico have been stamped by government officials as false. Cattlemen anticipate exportation of 500,000 animals annually to the United States. It has been suggested that Mexican meat packing concerns and the tanning industry in that southern republic are concerned about the proposed large-scale movement of live cattle to this country and the effect it will have on their operations which were greatly expanded after quarantine measures were applied to exports in 1946.

PRINCIPLES OF ANIMAL HEALTH

R. R. DYKSTRA, D.V.M.
Manhattan, Kansas

Shipping Fever in Cattle

So-called shipping fever of cattle is still a good deal of an enigma; at least this is the impression received in attendance at sessions of veterinary associations. The disease referred to in this article is that presumably due to the *Pasteurella boviseptica* or one of the group of hemorrhagic septicemia organisms. There is no unanimity regarding the specificity of this organism as the etiological factor in shipping fever of cattle. Scott and Farley (Department of Veterinary Medicine, Kansas Agricultural Experiment Station) in 1932 reported the isolation of this organism in 80% of the cases of shipping fever, and in 60% of the 23 cases examined found *P. boviseptica* "was associated with either colon type organisms or with members of the Alcaligenes group, usually *Alcaligenes bronchisepticus.*"

In regard to attempts to produce at least a degree of resistance to this disease, the report of the Committee on Miscellaneous Diseases of the U. S. Livestock Sanitary Association (*Proc. 53rd Ann. Mtg.*, 1929, p. 466) states that, "little or no immunity is conferred by bacterins or aggressin on animals in the incubation stage of the disease." Moreover the same report states "Your committee recommends the immunization of feeder and stocker cattle with either of these agents (bacterin or aggressin) when treatment can be applied at least ten days before shipment. Their use on these classes of cattle while in transit, or within a few days after they reach their destination, should not be encouraged."

The findings of Farley, An Epizoological Study of Shipping Fever in Kansas (*Jour. Am. Vet. Med. Assn.*, 80:165 [Feb.], 1932) is in agreement with the U. S. Livestock Sanitary Association report. However, Farley does state that "treatment of a small number of cattle with hemorrhagic septicemia antiserum resulted in a reduction of losses to a small figure."

The writer's conception of the handling is as follows:

1. Bacterin advisable approximately ten days before shipment.
2. Hold excitement of cattle at a minimum during shipment and transit to final destination.
3. On arrival at the farm, give protection from inclement weather, especially cold, wet weather. Supply all the water the animals want, but feed sparingly for several days and bring up to full feed gradually.
4. Promptly isolate all cattle showing clinical manifestations.
5. Administer hemorrhagic septicemia antiserum at the very first evidence of the disease.
6. Sulfanilamide per os in doses of 1 gr. for each pound of live weight is recommended therapy during the first 24 hours. Most practitioners give one-fourth of the above dosage every six hours so as to maintain a fairly constant blood level of the drug. The second and third days, the dosage is reduced by one-half. Others prefer to use sulfathiazole, sulfadiazine, or sulfamerazine, the latter in one-half the recommended dosage. Still others prefer parenteral medication using sodium sulfamerazine intravenously for a single initial dose, followed by oral dosage. Since the advent of antibiotics, there are more and more favorable reports on the treatment following the intravenous use of terramycin in doses of 500 mg. every 24 hours in calves weighing under 500 lb., or 1 gm. for those over 500 lb. Two doses are said to be sufficient.

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In veterinary circles there is still a good deal of discussion about the proper time to vaccinate young pigs; before or after weaning. As nearly as we have been able to determine it seems that immunization with hog cholera antiserum and virus at approximately six weeks of age—usually pigs are weaned at eight weeks—is the best time as such vaccination confers a solid, lasting immunity. Concurrent infections frequently interfere with the immunizing process if vaccination is delayed until pigs weigh 75 lb.

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The etiological agent of infectious canine hepatitis is not air-borne as is the virus of canine distemper. However, the virus of infectious hepatitis may be transmitted by kennel utensils and by the hands of attendants. Urine of convalescents remains infectious for an extended period after apparent recovery. Therefore, affected animals and those recently recovered should be kept isolated.

VETERINARY PARASITOLOGY

A. O. FOSTER, B.A., M.A., Sc.D.
Beltsville, Maryland

The 1951 Report of the chief of the Bureau of Animal Industry, USDA, released in January, contains items on the diagnosis, treatment, biology, and economic importance of livestock parasites.

High pressure, jet spraying proved to be the most efficient method of applying rotenone for large scale, area control of cattle grubs. Mixed sarcoptic and chorioptic scabies of cattle was eradicated by two pressure sprayings (500 pounds), with 0.075% lindane at an interval of 12 to 14 days. One application of 0.12% was promising. Dipping in 0.06% gamma BHC for the eradication of sheep scab conferred residual protection for at least 90 days under circumstances of continuous exposure. Eradication of keds was achieved by dips and sprays containing 0.1 to 0.25% toxaphene.

Concerning internal parasites, it was noted that ferric arsenate may be superior to lead arsenate as an ovine taeniacide, although, experimentally, pure infections with the broad tapeworm were not observed to cause discernible injury.

A 1:12 mixture of phenothiazine and salt, given free-choice, failed to control parasitism in a herd of nursing goats and their kids. The poor showing of the regimen was ascribed to the rapid build-up of parasites in the kids, and it is suggested that the measure may still have merit for dairy goats, as tentatively suggested by earlier work. Pursuant to suggestions that the good effects of phenothiazine could be due in part to a growth-stimulating property of the chemical, experiments conducted with lambs and rats led to the conclusion that the good results are not due to such effect of the compound, but rather, to its well known action against worm parasites.

Confirmation is reported of the value of 0.0125% sulfaquinoxaline in feed as a suppressive of cecal coccidiosis of poultry.

In anthelmintic trials on dogs and cats, a sulfone was found to be promising as a taeniafuge, and further studies with toluene confirmed the safety and efficacy of 0.1 cc. (erroneously given as 0.5 cc. in the report) per lb. of body weight against ascarids and hookworms.

Notes of biological interest include an instance of the apparent transmission from sheep to cattle of the common psoroptic scab mite and the observation that the hatching of coccidial oocysts in cattle is achieved, at least in part, by assistance from the common microorganisms of the rumen.

On the economic side, experiments in Alabama indicate that calves raised in individual portable pens, of the type devised for the control of coccidiosis and other fecal-borne infections, remained healthier and gained faster than comparable calves reared in standard-type calf barns.

In Georgia, experimental pasture-grown pigs acquired fewer parasites than pigs on bare lots; both groups were fed a basic grain ration but the latter received alfalfa in addition to compensate for the lack of green forage. At Beltsville, Md., heavy infestation of swine with the red stomach worm, *Hyostrongylus rubidus* was found to cause bloody feces, retarded growth, and even death; one pig died with 557 worms, and four less exposed survivors averaged 38.5 lb. gain in eight weeks as compared with 60 lb. for littermate controls. Trichinosis was transmitted to swine by way of the feces of infected rats, suggesting that the droppings of rats around garbage feeding establishments are a probable source of swine infection.

The cropworm of turkeys, *Capillaria contorta,* was found to cause serious injury and death, most of the losses unfortunately occurring in the fall when birds are nearing market age.

Investigations of disease and death among Canada geese at the Pea Island National Refuge, North Carolina, revealed the etiological significance of gross internal parasitism.

Because of parasitic conditions, whole-carcass condemnations during the fiscal year under federal meat inspection included 1,197 cattle and calves, 972 sheep and lambs, and 972 swine, these representing from 1 to 2% of total condemnations for all causes. Livers of cattle condemned because of liver flukes and other parasites exceeded 400,000, or about 25% of the total condemnations of this organ.

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During 1951, numbers of all cattle and calves on farms increased 6,000,000 head.—*Agr. Sit.*

POULTRY PRACTICE PROBLEMS

W. R. HINSHAW, D.V.M., Ph.D.
Frederick, Maryland

Anthrax Again

In the March 1952 issue of VETERINARY MEDICINE, page 118, I discussed anthrax in birds. Since then I have found a reference concerning birds as possible carriers which embarrasses me because I said in the article that to the best of my knowledge buzzards have not been proved carriers. In 1912, Dr. Harry Morris, University of Louisiana, conducted a series of experiments on a number of carrion feeders to determine their role as disseminators of anthrax. (La. Agr. Exp. Sta. Bul. 136)

I believe that subject is important enough from the standpoint of dissemination to other animals, if not to poultry, that a brief review of Morris' report is appropriate. He found that buzzards fed on anthrax-contaminated meat did not shed anthrax bacteria in their feces in four to 24 hours after being fed. The organisms could however, be isolated from the crop and gizzard for 20 hours but not in 24 hours. They were never found in the intestinal contents. It is of interest that he mentioned the possible antibiotic action of *Bacillus pyocyaneus* as having a role in killing the anthrax bacilli in the intestines since *B. pyocyaneus* was present in large numbers in the intestines of the buzzards examined.

In experiments to determine the time that contaminated beaks and feet of buzzards yielded positive anthrax cultures, it was found that virulent bacilli could be isolated from both the beaks and feet for at least 48 hours. He also observed that it is the habit of buzzards to vomit a few hours after eating a meal of carrion flesh. Cultures of such vomited flesh yielded virulent anthrax cultures. From these experiments Morris concluded that the buzzard is a potential carrier of anthrax even though *Bacillus anthracis* does not survive in its digestive tract.

Similar experiments with chickens showed that anthrax bacteria are not destroyed in the digestive tract and may be voided in the feces of these fowl, fed anthrax infected meat, for as long as 48 hours. Virulent anthrax bacteria for guinea pigs were found in all parts of the digestive tracts of chickens, and the cecum of one bird contained large numbers of organisms. In no instance, however, could he produce symptoms in chickens by feeding. One hen was fed five agar slopes of *B. anthracis* daily for 36 days without producing symptoms.

Incidentally dogs were found by Morris to be very important disseminators. Organisms were shed in the feces for six days after feeding. It was impossible to produce anthrax in dogs, even when virulent spores were fed daily for 90 days. Hogs carried the spores for five days, cats for four days, and opossums for four days.

Two more Louisiana bulletins by Morris dealing with insect transmission of anthrax are Louisiana Agricultural Experiment Bulletins 163 and 168 published in 1918 and 1919, respectively.

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Storrs Agricultural Experiment Station, University of Connecticut, has been granted $10,000. annually for three years to study encephalomalacia (crazy chick disease). This grant represents contributions from 42 feed manufacturers in 23 states. The studies will be conducted jointly by Dr. E. L. Jungherr, head of the Animal Pathology Department; Dr. E. P. Singren, Poultry Department and Dr. L. Matterson, Animal Husbandry Department. Encephalomalacia causes a high mortality rate among chicks. The cause is not known, but there is some indication that it is associated with a deficiency of vitamin E.

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The prerequisites for obtaining and keeping a breeding flock of chickens or turkeys free from pullorum disease are: (1) to establish a source of pullorum disease-free eggs; (2) to have such eggs hatched in a hatchery which accepts eggs only from pullorum disease-free flocks, and (3) to brood and rear the chicks and poults in brooders with equipment that has had no contact with birds of any species that have had pullorum disease.

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Marketing Farm Poultry (USDA Farmers Bulletin 2030) discusses a number of items of interest to veterinarians. Included are sections on preparing live poultry for market; processing poultry; grading and inspection of poultry; and packaging processed poultry. The bulletin contains a number of tables giving specifications for various standards and is well illustrated.

PUBLIC HEALTH

JAMES H. STEELE, D.V.M., M.P.H.
Atlanta, Georgia

Bovine Tuberculosis Nonexistent in Finland

A recent report from Finland on the prevalence of bovine tuberculosis indicates that the disease is no longer present in that country. This report was submitted to the *Journal of the American Medical Association* in 1951. Following are excerpts from it.

"It is probable that this disease was first introduced into the country a century ago with the import of diseased cattle from abroad. In 1891, tuberculin testing of cattle was started, and in 1894 it was found that 25% of 2,219 cattle gave a positive reaction. By 1897, more than 10,000 cattle had been tested, of which 18.6% gave a positive reaction. Bang's system of isolating positive reactors was adopted, at great cost to owners. In 1904, compulsory measures were introduced for stamping out the disease, and Bang's system was adhered to on the assumption that it would soon prove completely effective. However, between 1916 and 1928 the decline in percentage of positive reactors was disappointingly slow; it ranged between 4% and 7%. The adoption in 1923 of compulsory control of slaughtered cattle led to a disquieting and, at first, inexplicable discovery. In this year only, 0.15% of the cattle examined post mortem showed signs of tuberculosis, whereas 8.5% of the tuberculin-tested cattle gave a positive reaction. Many positive reactors were found at autopsy to show no other sign of tuberculosis, and both veterinary surgeons and owners came to distrust the tuberculin test as an effective aid to the eradication of bovine tuberculosis.

"At this stage a veterinary expert, R. Stenius, was called on by the Finnish Ministry of Health to investigate this curious disproportion between tuberculin and postmortem findings, and in 1928 he set to work on his task. In one herd he found only three positive reactors. They were calves, one of which he was allowed to slaughter. The postmortem findings were almost negative, only a few lymph nodes being swollen. Inoculation tests of experimental animals ultimately yielded tubercle bacilli of the human type. This observation led to the discovery that the calf had been in the care of a person who had subsequently died of pulmonary tuberculosis. This clue has led to a whole series of observations associating positive tuberculin reactions in cattle with open tuberculosis in human beings tending them. It was found that when a tuberculous person had been in contact for some time with a hitherto tuberculin-negative herd, some of its members, calves in particular, began after a few months or years to give positive reactions, usually slight. Such positive reactors showed no macroscopic evidence of tuberculosis, and they tended to become tuberculin-negative some time after contact with a tuberculous person had ceased.

"The avian type of tubercle bacillus can also render cattle tuberculin-positive. In such cattle there develops either an inactive and latent tuberculosis or an active form of the disease with the development of definite foci. Veterinary surgeons are now applying tuberculin tests designed to distinguish among the bovine, human and avian types of infection. A test indicative of a bovine infection calls for radical measures, whereas the demonstration of a human type infection requires only the removal of the infecting person, the cow in question being allowed to eliminate the infection in her own good time. Demonstration of the avian type should lead to discovery and slaughter of tuberculous fowls.

"Although infection of cattle with the bovine type has now been stamped out in Finland, cattle are still being tested with tuberculin, partly on the chance that infection with the bovine type may crop up again sporadically in cattle, and partly because tuberculin testing of cattle has proved to be an effective means for discovering hitherto undetected cases of infection of human beings with the human type of bacillus."

It is interesting to note that in the United States there have been a number of similar instances since the end of the war where the identification of cattle reacting to the tuberculin test revealed human infection on the premises or among those consuming milk from infected animals. Recently a case of bovine tuberculosis was identified in a Connecticut housewife who had no history of exposure to cattle. The number of human cases of bovine tuberculosis which are confirmed in the United States is becoming increasingly rare and will probably eventually disappear as they have in Finland.

ABSTRACTS OF LITERATURE

Control of Lungworm Infection in Sheep with Iodine

Treating lungworm infection of sheep with iodine-containing preparations, such as Lugol's solution, is not new; this treatment is commonly employed in Russia. More recently, research workers at the Veterinary College of Hannover, Germany,[13] found that a 2% colloidal iodine suspension gives excellent results when used for the removal of the large lungworm, *Dictyocaulus filaria*, and good results for the small lungworm, *Synthetocaulus spp.*

The suspension is given as an intratracheal injection in doses of 3 to 8 cc. for lambs, 12 cc. for yearlings, and 15 cc. for mature sheep. Dosages must be reduced to three-fourths or two-thirds of these amounts when weak or very sick animals are treated. The treatment may be repeated at one- to two-week intervals, if indicated. Of more than 3,000 sheep used in the experiment, only two showed spasms and breathing difficulties. Coughing, when observed, was of short duration.—*R. S.*

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Interpretation of Brucella Infection Tests

German veterinarians are of the opinion that the interpretation of agglutination values is at present not in accordance with the true facts of brucellosis[14]. It is suggested that agglutination findings of 26.5 to 49.4 should be evaluated not as suspicious, but as positive, and values of 10.6 to 26.5 as suspicious. This change is to be made when the Diernhofer method is applied. Blood samples with doubtful agglutination and with traces of agglutinin must be interpreted more carefully in future, or it will not be possible to eradicate this disease. The agglutination test is most dependable for cattle, less for swine, and least for sheep and goats.—*R. S.*

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More evidence is being made available that the common dog flea is an important vector of canine filariasis.

[13]Behrens, H., Die Behandlung des Lungenwurmbefalles der Schafe mit Merckjod. *Tierärztl. Umschau*, 6:394 (Nov.), 1951.

[14]Karsten, Prof. Dr., Ueber die Notwendigkeit, die Beurteilung der Ergebnisse der Serologischen Blutuntersuchung auf Brucella-Infectionen bei den Haustieren zu ändern. *Deutsch. Tierärztl. Wschr.*, 58:336 (Nov.), 1951.

Bovine Nutrition and Trace Minerals[12]

The rumen of cattle and sheep gives nutritional assistance due mainly to the organisms present. The particular activity of the rumen is the digestion of fiber or cellulose, the production of high quality protein, the building up of a whole galaxy of B-complex vitamins and the production of hormones. It has been found that every 1% increase in fiber above a level of 8% depressed the productive value an average of 2.5%. The rumen organisms require minimum amount of starch or sugar, nitrogen, certain minerals such as cobalt, and possibly certain fat-soluble vitamins. If there is doubt that the animals are getting enough of all the minerals they should help themselves to trace mineralized salt and steamed bonemeal or other suitable phosphorus carrier.

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Penicillin Treatment of Actinomycosis

Conservative treatment of actinomycosis of cattle is based primarily on the injection of iodine solutions; in many cases, it must be followed by surgery. This complicated treatment can be simplified by the use of penicillin-procaine in oil. A single dose of 600,000 to 900,000 units was used successfully[16] for the treatment of each of 21 affected cattle. Surgery was not necessary in any of the cases. The antibiotic is injected into the tumor mass.—*R. S.*

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Minerals in Ahimal Nutrition[13]

The feed of animals should contain about 4% minerals. Fourteen elements are essential: calcium, phosphorus, magnesium, chlorine, sodium, potassium, sulfur, iron, copper, cobalt, iodine, manganese, zinc, and fluorine. At least 12 others are found in normal animal tissue. Of the 14 listed above, only eight, in addition to salt, need be added to rations. These are calcium, phosphorus, iodine, copper, cobalt, iron, manganese, and fluorine. The proportion or ratio that one mineral element bears to others is extremely important.

[12]Bohstedt, G., *Feedstuffs*, 22:35, 1950, *Abs., Bio. Abs.*, 26:2F (Jan.), 1952.

[16]Teuscher, R., Ein Beitrag zur Behandlung der Aktinomykose des Rindes. *Tierärztl. Umschau*, 6:398 (Nov.), 1951.

[13]Bethke, R. M., *Feedstuffs*, 22:28, 1950, Abs. *Bio. Abs.*, 26:2F (Jan.), 1952.

Rupture of the Urethra Due to Faulty Emasculation

F. M. BEAM, D.V.M., Manhattan, Kansas

The owner of four Hereford bull calves presented them, stating that 36 hours previously they had been clamped by a high school vocational-agricultural (vo-ag) teacher for class demonstration. The calves were off feed and appeared dull and listless.

Symptoms

Large areas, 6 to 12 in. in diameter on the ventral surface of the abdomen, were swollen, cold, and edematous. Rectal temperatures were 102 to 103 F. Pulsation of the urethra was evident. Marks on the scrotum of each calf (see illustration) indicated extremely high

clamping with no attempt at staggering. The entire scrotum on one animal was cold and darkened. Improper application of the emasculatome, with rupture on the urethra, was the apparent cause.

Treatment

Calves were cast and prepared for surgery. Skin incision was made about 8 in. below the ischial arch on the median line. The penis was then brought to the outside by breaking through tissue with blunt dissection. Dorsal artery was isolated from the body of this organ; then the urethra and penis severed. For a distance of 1 in., the urethra was split longitudinally and four umbilical tape, interrupted sutures were used to secure it to the edges of the skin incision. About ¼ in. of the urethra was left to hang free, thus eliminating some of the possibility of strictures. No other suturing was attempted and the remainder of the area was left to heal as an open wound. One million units of penicillin were given as a prophylactic agent. The swollen area on the ventral surface of the abdomen was incised to allow drainage of accumulated urine.

The following day another patient was presented with similar symptoms and was treated in the same manner. The owner stated that the treated animals were sucking again and appeared to be normal. He was advised to market them as soon as the operative area healed.

A report four weeks following surgery stated that two of the calves recovered without complications. Three lost considerable weight and extensive sloughing occurred on the ventral surface of the abdomen. All animals recovered.

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Toxaphene Poisoning in Dogs

JOHN R. NUNN, D.V.M., Kerrville, Texas

At 8:30 a. m., February 12, 1952, the writer responded to an urgent call from a local Border Collie kennel owner. The night before, he had removed all dogs from the runs and sprayed the houses and the runs liberally with toxaphene spray using approximately ½ gal. of the condensed mixture to 50 gal. of water, or approximately 0.62% strength. He then replaced the dogs and retired for the night.

The next morning he arose to find three dogs dead, one grown bitch and two puppies. Nine other puppies, ranging in age from two to five months, exhibited symptoms of acute poisoning. Thus, of the 14 dogs occupying the runs, 12 were poisoned and only two were unaffected.

Symptoms varied in intensity. Some were in a position of opisthotonos with salivation, hyperesthesia, and nystagmus; while others appeared weak, were paddling, and either blind or oblivious to their surroundings. The remainder were generally nervous and apprehensive. Facial tics and profuse salivation were observed in all. Those having convulsions were cyanotic. Any sudden noise greatly intensified the spasms. Symptoms described here varied from time to time with the individual. These symptoms agree closely with those described for livestock poisoned by toxaphene[1].

All these puppies showing nervous symptoms were given nembutal®[1] intraperitoneally, vary-

®[1]Abbott Laboratories, North Chicago, Ill.

ing from 1 to 3 gr., depending on the size of the dog. Then all were washed thoroughly with tide,®[2] rinsed, and dried[3]. Following this, they were each given from 250 to 500 cc. of a 10% glucose in saline solution subcutaneously, divided between two or three sites.

For the next 24 hours the patients were watched closely, and sedation maintained by light intraperitoneal injections of nembutal. After the first 18 hours no nervous manifestations were in evidence and the puppies were beginning to show normal response.

Twenty-four hours later most of the puppies were able to drink warm milk and water, but were still weak, nervous and showed slight facial tics. They were then placed in a dark room and not disturbed for the next ten hours; after which all were apparently normal and were discharged.

The spray had settled in depressions in the concrete runs, which resulted in many small puddles. Poisoning resulted from drinking the solution or absorption through the foot pads and other skin areas.

REFERENCES

1. Radeleff, R. D., Toxaphene Poisoning: Symptomatology and Pathology. *Vet. Med.*, 44:436 (Oct.), 1949.

2. Radeleff, R. D., Practical Insecticide Toxicology for Veterinarians. Proceedings Book, Am. Vet. Med. Assn., 87th Annual Mtg., Aug. 1950. p. 68.

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Esophageal Worms in a Hound

P. W. TEDDER, D.V.M., Birmingham, Alabama

According to Mönnig, the esophageal worm, *Spirocerca lupi*, is common in most warm countries. Infection results in tumor formation in the walls of the esophagus, stomach, aorta or, more rarely, parasites are free in the stomach of dog, fox, wolf, and jackal.

Life Cycle

Eggs are passed through openings in the nodules or tumors, and eliminated in the feces of the infected host. These do not hatch until ingested by an intermediate host; any one of several species of dung-eating beetles. After hatching in the intermediate host, the larvae develop to the third stage and frogs, snakes, lizards, or birds may eat infected beetles and serve as transport hosts for infective larvae[1].

The dog becomes infected by ingesting one of these intermediate or transport hosts. The encysted parasites are liberated by the action of the gastric juices. They then penetrate the gastric wall, migrate in the walls of the gastric artery to the thoracic aorta, and thence to the walls of the aorta or esophagus, where they form nodules or tumors.

Typical history includes the fact that the dog has been eating well, but is losing weight. Vomiting immediately after eating is a common symptom.

Case Report

On July 5, 1951, a Black-and-Tan Hound was presented with the following symptoms: general flesh and apparent condition good;

Tumorous growth containing adult worms of *S. lupi* incised. The larger of these opened into the lumen of the esophagus.

dullness; tucked up abdomen; dyspnea; slight congestion of the mucous membranes of the eyes and oral cavity; palpation of the thoracic and abdominal cavities negative. While being examined, the patient vomited but passed only a mass of white mucus. Temperature and pulse were normal. Examination for endoparasites, ectoparasites, and heartworms was negative, as was urinalysis and a fluoroscopic examination. Tentative diagnosis: esophageal worms; possibly foreign body or tumor. Prognosis, unfavorable.

Treatment

The patient was hospitalized and given an enema and metamucil® in an effort to stimulate peristalsis. Vomiting continued after each meal for four to five days, in spite of repeti-

®[2]Procter & Gamble, Kansas City, Mo., detergent.

®G. D. Searle & Co., Chicago, Ill.

tion of this treatment daily. At this time, the dog began to gag and vomit continuously.

A stomach tube was passed with ease, suggesting freedom from stricture. With this in mind, exploratory laparotomy was recommended in anticipation of locating the causative agent in the vicinity of the stomach. No pathology of any type could be found in the cavity. The writer, having had experience in the past with what was called spasms of the cardiac valve of the stomach, massaged the cardiac and pyloric portions of the stomach to induce return of tone to these structures.

For the following ten to 12 days, the animal ate well and retained all food. Diagnosis was changed to sporadic spasms of the cardiac and pyloric valves with a favorable prognosis passed on to the owner.

A few days later, however, the dog started vomiting. Repeated examination of fecal specimens failed to reveal parasitic ova. (Eggs are small and do not float on most flotation media.)

The dog's weight having dropped from 40 lb. to approximately 20 lb., euthanasia was recommended.

Postmortem Finding

Only the thoracic cavity was examined. Four very large cysts and a number of smaller ones were found in the wall of the esophagus, approximately 1 in. from the diaphragm. Two of the larger contained 36 adult worms.

REFERENCE

1. Morgan, Banner Bill and Hawkins, Philip A., Veterinary Helminthology, Minneapolis, Minn., Burgess Publishing Co., 1949.

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Canine Pathology: An Historical Essay

(*Continued from page 245*)

ago by the use of the human eye (the direct method of observation), and without the armamentarium of diagnostic aids available now. There is still an enormous scope in work on natural animal diseases, for this same technic to be used in clinical medicine and pathology. In going back into history and showing respect for what was accomplished, we must nevertheless beware of an all too faithful subservience to authority. For, as the writer has pointed out elsewhere, in many fields this has retarded progress, and indeed, an excellent example is in the case of distemper-like diseases of dogs.

Calcium Deficiency in Newborn Lambs

H. L. NEWKIRK,* Stafford, Kansas

On December 14, 1951, a flock of 148 ewes of mixed breeding, began lambing. All appeared to be in good condition and none had exhibited any signs of illness.

Four of the first ten ewes delivered twins and one bore triplets. One twin in three of the four pairs was unable to stand. Lambs appeared normal and were alert. All of them would nurse if held up to the ewe. The only symptom observed was weakness and inability to stand.

By questioning the owner, it was learned that for the past two months the ewes had ranged on combination wheat and rye pasture. The pasture at this time was well picked down, being short and dry. In addition, they were fed fine, leafy, sorghum fodder, with good heads, every morning; third-cutting, only fair, alfalfa hay every night. Salt and a mineral mixture were available free choice.

Diagnosis was withheld, but the possibility of calcium deficiency was suggested.

Treatment was begun immediately on the four lambs. Feeding by bottle, 2 oz. of milk with 1 ts. of calcium carbonate added, was given every four hours. Two lambs were able to stand and nurse after the third feeding. The other three made apparent recoveries in 24 hours or less.

Ewes were put on a diet of good, leafy, green alfalfa and a mixture of milo and oats with calcium carbonate added. Two more weak-kneed lambs were dropped; both in multiple births, during the following two days. No further difficulties were experienced.

Due to the rapid recovery on the calcium supplement, and the relief of symptoms after addition to the ration of the ewe, it is reasonable to assume a calcium deficiency was responsible and that deficiency disease may occur in newborn lambs without manifesting itself in the ewe.

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The indiscriminate use of antibiotics in the form of intramammary infusions when infection has not been established is to be discouraged. It is contended that such a practice has made many gallons of milk unfit for the manufacture of cheese and cultured milk. In instances such as this, the antibiotic residue in the milk inhibits, or at least affects, the cultures added to the milk in the making of these commercial dairy products.

*Senior student, class '52, Kansas State College, Manhattan.

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BOOK REVIEWS

Books reviewed in this department and any other books, if available, may be obtained by remitting the published price to the Book Department, Veterinary Medicine, Suite 803 Livestock Exchange Building, Kansas City 15, Missouri.

AN OUTLINE OF THE INFECTIOUS DISEASES OF DOMESTIC ANIMALS, by *Ival Arthur Merchant*, D.V.M., Ph.D., head of Department of Veterinary Hygiene, Iowa State College, Ames. 356 pages. Burgess Publishing Co., Minneapolis, Minn., publishers, 1951. — Price $5.00.

This volume, a first edition, was created from a systematic outline of lecture notes for senior students on the important diseases of livestock (except poultry) and will serve as a reference book to obtain, in concise form, information important in making diagnoses.

Each disease is described in brief under the following headings: synonyms, definition, history, etiology, epizootiology, symptoms, pathology, diagnosis, prognosis, treatment, prophylaxis, state and federal regulations, public health relationships, references for study.

Fifty-eight specific diseases are treated, according to the Outline, in short concise statements; every line worth reading should a particular infection occur in your territory. To illustrate, the subject of anthrax is covered in eight pages.

A splendid library reference book for practitioners' use, rather than a bacteriological laboratory manual. The author omitted X disease in cattle, rhinitis, edema, anaplasmosis-like diseases in swine, and canine hepatitis. The reviewer anticipates they will be included in the next edition.

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STANDARD VALUES IN BLOOD, edited by *Errett C. Albritton*, A.B., M.D. Fry Professor of Physiology, The George Washington University, Washington, D. C. Prepared under the direction of the Committee on the Handbook of Biological Data, American Institute of Biological Sciences, The National Research Council, United States Air Force, Wright Air Development Center, Wright-Patterson Air Force Base, Dayton, Ohio. To be issued through regular publishing channels later this year. Availability and price to be announced.

Material is presented exclusively in tabular form. Standard values in blood for man and other vertebrates are given, including domestic livestock, cattle, swine, sheep, horses, dogs, cats, goats, and rabbits. Values are arranged under sections. These include physical properties, coagulation phenomena, blood groups, erythrocytes and hemoglobin, leukocytes, blood and bone marrow cells, water, carbohydrates, lipids and proteins, amino acids and nonprotein nitrogen compounds, phosphorus and sulfur compounds, vitamins, hormones, enzymes, electrolytes, minerals, gases, acid-base values, effects of radiation and storage, and effective blood levels of therapeutic agents. In brief, it presents comprehensive and complete data on blood, which is reliable. No less than 600 investigators in biology and clinical medicine have authenticated the data.

Tables are unique in their treatment of well known phenomena of biological variability. In addition to giving a single representative value for each item covered in any table, these present information of variability in a usable form.

An extensive bibliography is appended which also contains the name of the contributor for each value.

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FILMS

Striking Back Against Rabies: Shows activities of various public health organizations. Deals primarily with quarantine, emergency inoculation clinics, participation of veterinarians and health officers, laboratory diagnosis, human treatment and publicity. Description; 16 mm, B & W, sound, time 12 min.—Available for free loan for professional training by the Audio-Visual Production Services, Communicable Disease Center, Public Health Service, Atlanta 3, Ga.

Laboratory Diagnosis of Rabies: Step by step procedure in rabies diagnosis by identification of Negri bodies. Explains mouse inoculation tests. This film details the brief sequences on laboratory diagnosis in Striking Back Against Rabies. Description; 16 mm, B & W, sound, time 5 min.—Address Communicable Disease Center, Public Health Service, Federal Security Agency, Atlanta 3, Ga.